CHRONIC LUNG DISEASE IN AUSTRALIAN ABORIGINES

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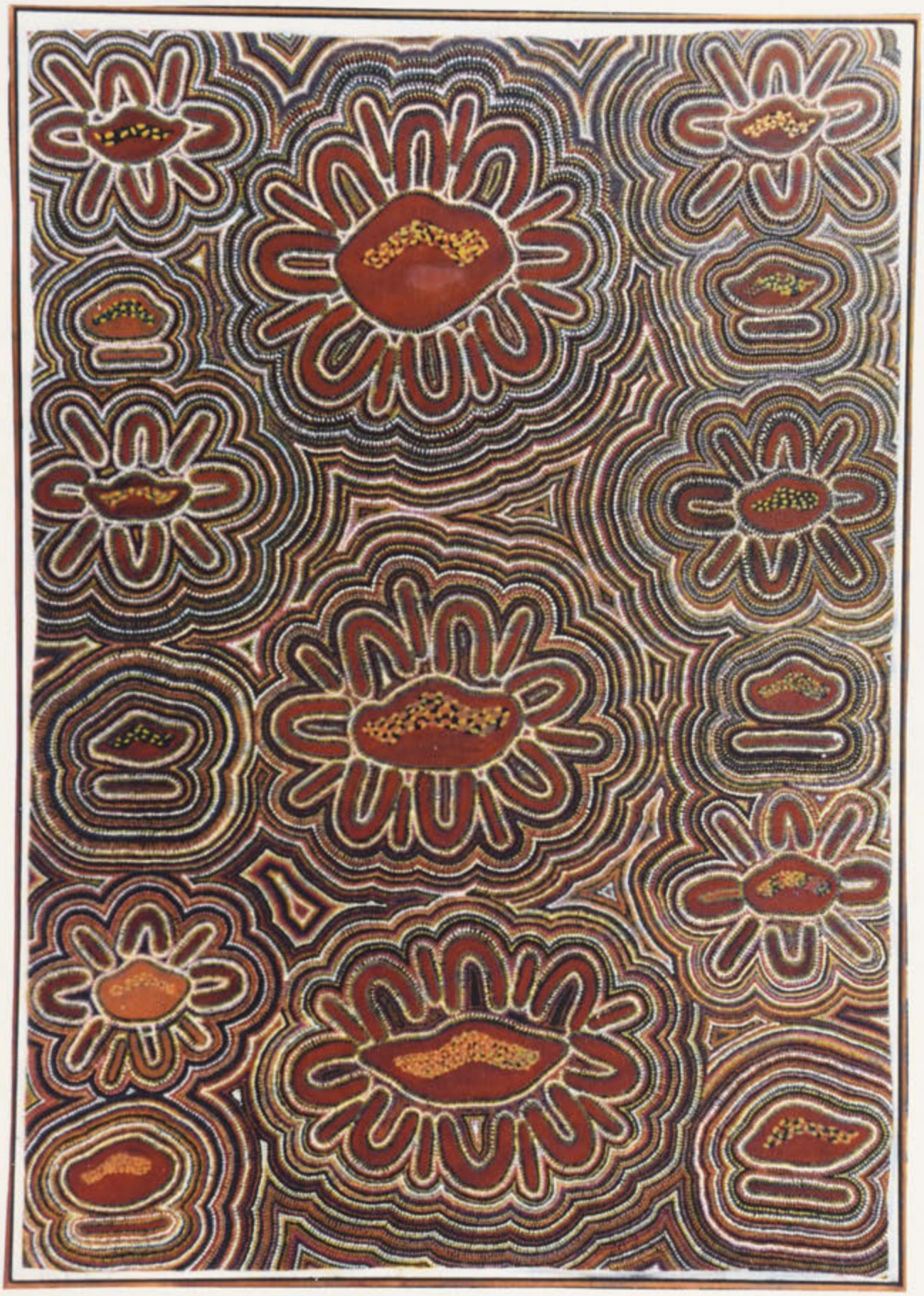

"Bushtucker" by Lena Pwerle 1992*

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Declaration

Except where otherwise indicated, this thesis is the result of my own work carried out while I was a PhD student at the National Centre for Epidemiology and Population Health (NCEPH) at the Australian National University in Canberra.

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Abstract

This thesis describes a study of 1,252 Aborigines who were living in 4 rural communities, two in Cape York and two in central Australia. The aim was to define normal lung function and to determine the prevalence of and risk factors for current asthma, bronchial hyperreactivity (BHR) and chronic lung disease. The participation rates ranged from 51 to 87%. Current asthma was diagnosed in subjects with a history of wheezing in the last 12 months and coexisting BHR ($PD_{20}FEV_1 < 3.9$ micromols of histamine). Chronic lung disease was diagnosed if a subject's FEV_1, FVC or the FEV_1/FVC ratio was greater than two standard deviations below that in a reference population of 593 participants without signs or symptoms of lung disease. Subjects with chronic lung disease and an abnormal FEV_1/FVC ratio were diagnosed as having chronic obstructive pulmonary disease (COPD) and those with a normal ratio were diagnosed as having non-obstructive lung disease.

The prevalence of smoking ranged from 1-78% in women and from 51-75% in men. "Knowledge" about the health consequences of smoking was poor. In one community the prevalence of self-reported petrol sniffing was 44% among 15-25 year old males and 33% among females.

The prevalence of asthma among adults was low in comparison with non-Aboriginal Australians and in children asthma was almost non-existent. The only significant risk factor identified for asthma was allergy to cats. The prevalence of BHR among adults was similar to that in non-Aboriginal Australians but in children the prevalence was low. The risk factors for BHR were house dust mite allergy, feline allergy and cigarette smoking. The low prevalence of asthma in the children may be secondary to a reduced atopy acquisition rate.

The values predicted for FEV_1 were approximately 20% lower than those predicted for Caucasians of the same height, age and gender. In contrast the values predicted for FVC were approximately 30% lower. Consequently the

FEV_1/FVC ratio was high in comparison with Caucasians. Asymptomatic cigarette smokers had higher predicted values of FEV_1 and FVC than non-smokers. The rate of decline of FEV_1 and FVC with age in healthy smokers and non-smokers appeared comparable to that observed in non-smoking Caucasians.

The prevalence of chronic lung disease in 7-19 year old males was 7.1% and among 20-84 year old males was 19.3%. The prevalence of chronic lung disease in females was lower: 2.2% among 7-19 year olds and 5.9% among 20-84 year olds. Respiratory symptoms and signs were not useful for identifying or characterising subjects with COPD or non-obstructive lung disease.

The prevalence of COPD was 11.8% in males and 4.5% in females. COPD was not detected in children. Ex-smokers were at increased risk of having COPD. Increasing age was an independent risk factor for COPD. The lifestyle in one "traditional" community was associated with an independent reduction in the risk of having COPD. Twelve percent of subjects with COPD had asthma. BHR was associated with COPD but the prevalence of BHR among those with COPD was low compared with non-Aboriginal people with this disease.

The prevalence of non-obstructive lung disease was 7% in males and 2% in females. Petrol sniffing and male gender were significant independent risk factors for non-obstructive lung disease. Age was not an independent risk factor for this abnormality. Subjects from the community with the most adequate housing were significantly less likely to have non-obstructive lung disease than subjects from communities where the housing was of a poor standard and where serious lower respiratory tract infections in childhood were said to be common.

The findings suggest that initiatives that lead to more Aboriginal people living in decentralised family groups in adequately maintained houses have the potential to reduce the burden of COPD and non-obstructive lung disease. The mechanisms by which these factors might influence the prevalence of chronic lung disease need further investigation.

Definitions and Abbreviations

CY1 Cape York 1	An Aboriginal community on the eastern seaboard of Queensland near Cooktown.
CY2 Cape York 2	An Aboriginal community on the western side of Cape York at a latitude north of Cooktown, Queensland.
CA3 Central Australia 3	An Aboriginal community several hundred kilometres north-east of Alice Springs in the Northern Territory.
CA4 Central Australia 4	An Aboriginal community several hundred kilometres south of Alice Springs in South Australia.
Regional variation	Variation between communities.
ATS	American Thoracic Society.
BTPS	Body temperature and pressure, saturated with water vapour.
FEV_1	Forced expiratory volume in one second (expressed in terms of BTPS).
FVC	Forced vital capacity (expressed in terms of BTPS).
Asthma	The combination of recent wheeze and bronchial hyperresponsiveness (BHR).
Atopy	A wheal reaction of 3mm or more to a skin test using standardised allergens.
BHR	Bronchial hyperresponsiveness, defined as $PD_{20}FEV_1$ less than or equal to 3.9 µmols of histamine.

Chronic cough	A history of daily coughing for more than two years.
CLD	Chronic lung disease was diagnosed if a subject's FEV_1, FVC or the FEV_1/FVC ratio was greater than two standard deviations below that in a reference population of 593 participants without signs or symptoms of lung disease.
COPD	Chronic obstructive pulmonary disease was diagnosed in people with CLD who had an abnormal FEV_1/FVC ratio.
NOLD	Non-obstructive lung disease was diagnosed in people with CLD who had a normal FEV_1/FVC ratio.
Crepitations	Crackling sounds on auscultation which may be associated with disease of the pulmonary parenchyma or fluid in alveoli.
DP	*Dermatophagoides pteronyssinus*
*Der P*1	*Dermatophagoides pteronyssinus* 1 allergen.
DF	*Dermatophagoides farinae*
Ever-smoker	A person who is a current smoker or an ex-smoker.
HDM	House dust mite.
HLA	Histocompatability locus antigen.
IgE	Immunoglobulin E
Loose cough	The quality of looseness (moistness) observed in a cough; correlates with productive cough.
MRC	Medical Research Council (British).
$PD_{20}FEV_1$	Dose of histamine required to produce a 20% percent fall in FEV_1 during a histamine challenge test.
Petrol sniffer	Person who regularly inhales petrol fumes. A form of substance abuse.
Recent wheeze	A history of wheezing in the last 12 months.
Rhonchi	A musical sound heard on auscultation of the chest which may be observed in asthma and other airway diseases.
SMR	Standardised mortality ratios.

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CHAPTER 1

Introduction

1.1 Introduction

Australian Aborigines are significantly more likely than non-Aboriginal Australians to die or be hospitalised as a result of respiratory disease. Little is known about the nature or causes of chronic lung disease (CLD) in Aborigines. The principal objective of this study was to document the nature and prevalence of CLD in four Aboriginal communities and to consider the role of several known risk factors for respiratory disease.

Respiratory health was assessed by determining whether subjects had chronic cough, recent wheeze, basal crepitations, basal rhonchi, loose cough, bronchial hyperreactivity or abnormal spirometric function.

As there were no published reference values for lung function in healthy Aboriginal children and only one set in adults (1) it was necessary to measure spirometric function in healthy subjects so that equations for determining reference values could be developed.

Issues related to cigarette smoking were explored because there is a belief that smoking is a serious public health problem in Aboriginal communities and because little is known about Aboriginal smoking (2). Data were also collected about tobacco chewing and alcohol consumption because in the author's experience these substances are commonly used in some Aboriginal

communities and because any associations between tobacco smoking and other substance use could be of public health significance.

It is readily apparent when one visits most Aboriginal communities that the prevalence of chronic cough and "fruity cough" (loose cough) are high. Health practitioners have no rational way of interpreting the significance of these observations because it is not known if they are associated with a specific respiratory disease. An important additional objective of the present study was to examine the associations between common respiratory symptoms and signs and CLD.

In the present study, the research process was used as an opportunity for Aboriginal health worker training and community health development. This was done because the National Health and Medical Research Council guidelines for the conduct of Aboriginal health research (3) require researchers to conduct their work in a manner that leads to tangible community benefits. The guidelines also emphasise the need for meaningful community consultation and participation in research. Because these guidelines are relatively new the research process is documented in detail.

1.2 Potential Risk Factors for CLD in Aboriginal Communities

1.2.1 Lower Respiratory Tract Infection

Before the age of three years many Aboriginal children commonly require repeated hospitalisation (4) for severe episodes of lower respiratory tract infection. In non-Aboriginal children lower respiratory tract infections have been shown to be associated with reduced forced expiratory volumes and reduced lung growth (5). Whilst it is well recognised that some Aboriginal children develop bronchiectasis as a consequence of early lower respiratory tract infection (6), it is not known if some infections are more subtly affecting lung growth and development. In the present study the author did not seek to relate specific episodes of lower respiratory tract infection to abnormalities of ventilatory function but rather sought to broadly examine the possibility they might be associated with the development of CLD.

1.2.2 Environmental Factors

Although many clinicians believe that substandard housing and crowding are associated with increased morbidity from respiratory disease, there is little "hard" evidence to support this notion (7-9). Living conditions for Australian Aborigines vary considerably from region to region. While some

live in adequately maintained housing, this is the exception (10). More often they live in poorly maintained or derelict houses, without access to running water or functioning showers and toilets (11). Many Aboriginal people live in shelters constructed from corrugated iron, branches or tarpaulins. Assessing the impact of housing and environmental factors on respiratory health is complicated because there are many potential confounders (12-14) and because the mechanism by which substandard housing and crowding might cause lung disease is unclear. Important factors associated with these conditions might be respiratory infections, dust and smoke exposure. The present study examined people from different regions thus providing an opportunity to consider the impact of different housing conditions on respiratory health.

1.2.3 Cigarette Smoking

Although cigarette smoking in Aboriginal people has been studied by several investigators (15-17) little is known about their patterns of smoking, their understanding of the health consequences of smoking, or the effect of smoking on their lung function (2). The present study afforded an opportunity to explore these issues. It was hypothesised that current smokers would have poorer lung function than non-smokers, that health knowledge would be low in comparison with non-Aboriginal Australians and that there would be a significant relationship between health knowledge and current smoking status.

1.2.4 Asthma

Asthma is an important health problem for non-Aboriginal Australians (18) and may be associated with fixed abnormalities of lung function and atopy to common aero-allergens. The few reports that refer directly or indirectly to the prevalence of asthma in Aborigines provide varying estimates for its prevalence (19-28). Anecdotal reports from Cape York by Dr John Thompson, Thoracic Physician, Cairns Base Hospital, suggested that the prevalence of asthma in adults and children was low (0 to 5%). Dr Thompson also observed regional variation in the prevalence of asthma in these areas. The present study allowed the first formal documentation of the prevalence of asthma in Aboriginal adults and children.

Evidence has been accumulating that house dust mite allergy and asthma are causally associated (29). The prevalence of house dust mite allergen exposure and its relationship to asthma in Aborigines has not been investigated previously. In addition, the importance of atopy to other common aero-allergens has not been assessed in Aborigines.

Bronchial hyperreactivity (BHR), an airway abnormality associated with asthma and chronic obstructive airways disease, can be detected by assessing the fall in FEV_1 that may occur following increasing doses of inhaled histamine (30). This procedure is known as a histamine challenge test. The test has proven reliability and has now been used extensively in epidemiological studies of asthma. There is only one report assessing bronchial hyperreactivity in Aboriginal children and there are none in adults. The present study provided an opportunity to study the prevalence and associations of BHR in Aborigines.

1.2.5 Genetic Factors

Dr Thompson hypothesised that the emergence of asthma in one Aboriginal community located on the north Queensland coast (identified as CY1 in this study) might be related to the influence of "non-Aboriginal" genes associated with asthma or atopy, introduced as a result of racial mixing. Professor Sue Serjeantson (Department of Human Genetics, John Curtin School of Medical Research ACT) has an interest in using the human leucocyte antigen system gene complex to study human migration and admixture in the pacific region. The present study afforded an opportunity to determine if there was any association between the presence of asthma (or atopy) and HLA subtypes not usually found in Aborigines.

1.2.6 Petrol Sniffing

In some Aboriginal communities children spend many hours each day inhaling fresh petrol vapour, so called petrol sniffing (31). It is well recognised that long term petrol sniffing may result in serious neurological and liver disease but it is not known if petrol sniffing is an important cause of CLD.

1.3 The Participating Communities

All the indigenous residents over the age of 4 years in four rural Aboriginal communities were invited to participate in the present study.

1.3.1 Cape York 1 (CY1)

CY1 had the longest history of contact with Europeans of the four communities studied and its residents generally lived in well maintained houses. The level of literacy and numeracy was relatively high and English was the principal language. The living conditions in CY1 were more westernised than those in the other three communities. Lower respiratory tract infections in children were said to be uncommon but asthma was reported to be prevalent. Data from this community were examined to see if there was any

evidence that these apparently healthy living conditions were associated with a prevalence of CLD that was lower than that in the other three communities.

1.3.2 Cape York 2 (CY2)

CY2 is more isolated than CY1, the standard of housing at the time of the present study was less adequate and alcohol abuse was also a serious problem. The community comprised two main tribal groups that lived together uneasily. Tior and Munkin were the dominant languages. Lower respiratory tract infections in children and asthma were said to be uncommon but CLD was thought to be prevalent.

1.3.3 Central Australia 3 (CA3)

CA3 was the most "traditional" of the four communities and its members lived on a series of decentralised "outstations" or "homelands". As a consequence there was no central community. Housing ranged from simple shelters to free standing simple dwellings with running water and ablution facilities. Alyawarra and Anmatyerre were the main languages. This was the only community in the study not to have been a mission and significant contact with Europeans did not occur until the 1920s. It has been reported that childhood pneumonia although prevalent in this community was rarely serious enough to require evacuation to the regional hospital in Alice Springs (32). CA3 was selected to determine whether there was evidence that the traditional lifestyle in the community conferred some objective respiratory health advantages.

1.3.4 Central Australia 4 (CA4)

CA4 is a community created in the 1960s comprising several related tribal groups who spoke Pitjantjatjara and Yankunytjatjara. At the time of the present study the housing in CA4 was grossly inadequate and overcrowding was widespread (11). The author and his wife worked as medical officers in the community during 1986 and 1987. This study had its origins in that experience. The author observed that at least one episode of childhood pneumonia in the first two years of life was universal and that adult lung disease appeared common. Petrol sniffing was prevalent among teenage children in this community. CA4 was selected to determine if there was objective evidence that either the poor living conditions or petrol sniffing were associated with a high prevalence of CLD in comparison with the other communities.

1.4 Aims

In the four selected communities the aims were to determine:

- The prevalence of smoking and the nature of any associations with tobacco chewing and alcohol consumption;
- The level of awareness about the dangers of smoking;
- The prevalence of bronchial hyperreactivity, atopy and current asthma in adults and children;
- The relationship between house dust mite allergen exposure, house dust mite allergy and asthma;
- Whether asthma and atopy in Aborigines from Cape York are associated with "non-traditional" HLA alleles;
- Whether asthma and atopy in Aborigines are associated with elevations of Immunoglobulin IgE;
- Prediction equations for FEV_1 and FVC in "healthy" Aborigines;
- The prevalence of chronic cough, recent wheeze, loose cough and crepitations;
- The risk factors for chronic cough, loose cough and recent wheeze;
- The prevalence of chronic obstructive pulmonary disease (COPD) and non-obstructive lung disease (NOLD) in adults and children by region, age and gender;
- The likely risk factors for COPD and NOLD;
- The clinical features of COPD and NOLD.

1.5 Methods

All residents over the age of four, from each of the four communities, were invited:

- To answer questions about respiratory symptoms, alcohol consumption and smoking;
- To have measurement of their sitting height, standing height and weight;
- To be assessed for the presence of loose cough;
- To have auscultation of the posterior lung bases;
- To have their tidal carbon monoxide level measured;
- To have blood drawn for HLA class two antigen analysis and IgE estimation (selected subjects, Cape York only);
- To have house dust collected for house dust mite allergen load (selected subjects);
- To have spirometry performed (FEV_1 and FVC);
- To have a histamine challenge test performed;
- To have skin tests for atopy to common allergens.

1.6 Organisation of the Thesis

There are nine chapters that follow this introduction. In chapter two the studies that have relevance to Aboriginal respiratory disease are reviewed. In chapter three the ethical framework of the study, the history and current circumstances of the participating communities and the process of obtaining informed consent and feeding back the study's findings are summarised. In the next chapter the sampling procedure and details of participation are described.

The following five chapters present the findings regarding cigarette smoking, asthma and atopy, normal lung function, chronic and loose cough, COPD and NOLD. The thesis concludes with a chapter that highlights the important findings and addresses their public health implications.

This research was conducted in order to develop a better understanding of the constellation of factors that influence "Aboriginal Health". The author particularly wanted to explore respiratory health issues in a way that would produce data suitable for use as a health development resource for Aboriginal people and their health services. The author has drawn on the insights and experience he gained from living and working in CA4 in 1986-87 and from discussions with many people. It is his hoped that the thesis will provide impetus for Aboriginal programs that lead to healthy change.

CHAPTER 2

Literature Review: Respiratory Health and Disease in Aborigines

2.1 Introduction

This chapter highlights the significance of respiratory disease as a cause of death and hospitalisation among Aboriginal people. The studies that specifically address lung disease and lung function are also discussed in chronological order. The chapter concludes with a summary.

2.2 Mortality Studies

In 1985 Hicks (33) published standardised mortality ratios (SMR) for Western Australian Aborigines. In males the SMR was 2.4 times the total Western Australian rate and in females the SMR was 2.9. Diseases of the respiratory system (ICD-9 V111) were the second most prevalent cause of death for males, and the fourth most prevalent cause for females. Approximately half the respiratory deaths were due to pneumonia and half were attributed to chronic obstructive pulmonary disease (COPD) and emphysema (ICD-9: 492 & 496). The numbers were too small to calculate standardised mortality ratios for each disease.

In 1988, Plant (27) published an analysis of the 1,651 Aboriginal deaths that occurred in the Northern Territory (NT) from 1979 to 1983 inclusive. The total Australian population was used as the standard to calculate SMRs. Plant found the crude death rate in Aborigines to be 11 per thousand. The SMR for males

was 3.2 (95% CI 3.0–3.4) and females was 3.9 (95% CI 3.6–4.2). Respiratory and cardiovascular disease were the dominant causes of death after the age of 35. Plant calculated that the SMR for death from diseases of the respiratory system was 9 in males and 15 in females. The relative risk of death from pneumonia and influenza was 22 (95% CI 17–26) in men and 19 (95% CI 14–25) in women. The relative risk of death from COPD and asthma (ICD-9: 493) was 5 (95% CI 4–6) in men and 11 (95% CI 8–14) in women. Plant's study is the most comprehensive Aboriginal mortality study to date. It shows that the death rate from pneumonia and COPD (or some type of chronic lung disease) in Northern Territory Aborigines is significantly higher than in the non-Aboriginal Australian population.

The only other mortality study of significant size is that by Honari (34) who reported 967 Aboriginal deaths occurring in Queensland, Western Australia, South Australia and the Northern Territory in 1985. The study by Honari is not as powerful as that of Plant as it examined fewer deaths. However, the findings support Plant's work and indicate that the excess Aboriginal mortality from respiratory disease, identified in the Northern Territory, was not peculiar to that region.

Perinatal and infant mortality rates in Aborigines have declined in the last twenty years but are still 2 to 3.6 times higher than for non-Aboriginal Australians (28). Respiratory diseases in Northern Territory Aboriginal infants were the cause of 18.2% of infant deaths between 1979 and 1983. In the non-Aboriginal population respiratory diseases caused only 5.6% of infant deaths during this period (24). Thus, whilst it is true that at all ages death from respiratory diseases are more common in Aboriginal than non-Aboriginal people, most of the excess deaths from these diseases are occurring in adults rather than infants.

2.3 Hospital Admission & Discharge Studies

The most comprehensive Aboriginal hospitalisation study is that by Devaneson *et al* (1986) entitled *Health indicators in the Northern Territory* (24). Rate ratios (RR) for hospital separation were calculated by dividing the Aboriginal rates by the non-Aboriginal rates. The RR for hospital separations were highest among 0 to 4 year olds and 25 to 64 year olds (RRs >2.0). Respiratory diseases (ICD-9: 460-519) were the second most prevalent cause (16%) for hospital separations in Aboriginal men and the fourth in women (9%). Among non-Aboriginal people, respiratory diseases were also the second most prevalent cause of hospital separations in men (9%) and fourth in women

(6%). The Aboriginal male respiratory admissions comprised pneumonia 59%, upper respiratory tract infection (URTI) 11%, COPD 8% and asthma 3%. The Aboriginal female respiratory admissions comprised pneumonia 49%, URTI 13%, COPD 10% and asthma 5%. Devaneson *et al* showed that when compared with the non-Aboriginal population the proportion of respiratory admissions in Aborigines for pneumonia was relatively high and for asthma was relatively low. The proportion of respiratory admissions due to COPD was similar in the Aboriginal and non-Aboriginal populations. These data concur with the findings from the mortality studies cited above as they suggest that pneumonia and COPD are the prevalent respiratory problems in Northern Territory Aborigines.

2.4 Epidemiological Studies

In 1967 Gandevia (22) reported findings from Papunya, an Aboriginal community in the Northern Territory with a population of approximately 800 people. Subjects were examined for loose cough, crepitations and rhonchi. Subjects were also questioned regarding recent chest infections (defined as any chest infection in the preceding two and a half years that required antibiotics). The chest infection history was validated by examining the community's hospital/clinic records. The community was free of industrial air pollution and cigarette smoking. Gandevia's study was the first population based study of respiratory disease in an Aboriginal community. The study had added interest because within the community there were three distinct Aboriginal groups. The participation rate among children was approximately 75% and among adults was 17%.

Gandevia found the prevalence of recent chest infection in all three tribal groups was high (range in children 27-50%, adults 22-85%). The prevalence of loose cough was also high (range in children 50-79%, adults 28-79%), and basal crepitations were prevalent (range in children 24-65%, adults 11-71%). The findings highlight the impact respiratory tract infections may have in small disadvantaged communities. The prevalence of chronic lung disease at the time of the study remains unknown because tests of pulmonary function were not performed. The significance of the basal crepitations is also unknown because repeat examinations and CXRs were not performed. It is noteworthy that rhonchi were not detected in any children as it suggests that the prevalence of asthma was low.

In 1968 Maxwell *et al* (35) published the results of a study performed in Papunya. Maxwell *et al* X-rayed 271 of the 329 children obtaining postero-

anterior views of their chests. They also examined the children for loose cough and auscultated the bases of their lungs. Maxwell *et al* found that 21% of male children and 33% of females had either a loose cough, crepitations or rhonchi (not cleared by coughing). These observations are consistent with those of Gandevia. He also noted that 27% of boys and 8% of girls had either signs of chronic lung disease (chest wall deformity, clubbing or impaired chest movement) or acute lower respiratory tract infection. It is unclear what proportion of children had signs of chronic lung disease without signs of acute lower respiratory tract infection. The predominant radiological abnormalities were bronchiectasis of the left lower lobe 1.8%, probable bronchiectasis 3.2%, accentuated basal shadows 3.6% and pulmonary consolidation 16%. Although the prevalence of these abnormalities in non-Aboriginal Australians is unknown, most medical practitioners would probably expect them to be significantly lower than that found by Maxwell *et al*. Although some of the children with consolidation may not have had pneumonia (because half of them had no signs of lung disease) the study still provides compelling evidence that the prevalence of pneumonia was high. This remains the only published (population based) radiological study of Aboriginal children. It showed that the prevalence of pneumonia and bronchiectasis in Papunya was high and that loose cough and crepitations were also prevalent.

In 1972 Maxwell (6, 36) investigated 118 Aboriginal children from communities around Alice Springs who were thought to have chronic lung disease on clinical grounds. The investigations showed that 83 had bronchiectasis and 35 had chronic bronchitis (criteria for diagnosis not defined). Both groups of children gave similar histories. Sixty percent of the bronchiectatics reported symptoms since at least the age of two. Of the children with bronchiectasis, 47% had a history of bronchiolitis, 37% pneumonia and 11% recurrent chest infections. Fifty-five percent of those with bronchiectasis had been dehydrated at the time of at least one of these respiratory episodes. These illnesses remain prevalent in rural Aboriginal children (4). Most (84%) of the children with bronchiectasis reported chronic productive cough and on examination loose cough was universal. Twenty-eight percent of the children with bronchiectasis had no abnormality on repeated physical examination. None of the children had cystic fibrosis, hypo-gammaglobulinaemia or evidence of alpha-1-antitrypsin deficiency (37). Half of the children were said to have abnormalities of pulmonary function but the details were not reported. The anatomical distribution of the bronchiectasis was similar to that seen in non-Aboriginal children.

Maxwell opined that bronchiolitis, particularly if associated with dehydration, was an important cause of chronic respiratory disease in Aboriginal children. He also asserted that chronic lung disease in Aboriginal children was a result of environmental rather than genetic factors. He observed that most children with chronic lung disease lived in simple overcrowded shelters with constant opportunity for cross-infection. Maxwell's paper is valuable because it describes the features of a selected group of patients with chronic lung disease and forwards some hypotheses regarding aetiology.

In 1973 Moodie (21) observed there were no studies of asthma in Aborigines. He studied 320 Aboriginal children living in a New South Wales coastal community and documented a history of wheeze in 3.4%. He also observed that on examination "very few" of the children had signs of asthma. The prevalence of wheeze was low when contrasted with Peat *et al* who found that 24.2% of 1,668 non-Aboriginal children aged 8–10 years had wheezed in the previous 12 months (38). Moodie concluded that in the community he examined the prevalence of asthma was less than 4%.

In 1975 Hiller reported a one year survey of chest X-rays performed in the Alice Springs Hospital on children aged one month to 14 years (39). An attempt was made to distinguish between "full blood" Aboriginal and "part Aboriginal and Caucasian" children. The X-rays were taken in both the antero-posterior and lateral positions. A total of 988 X-rays were reported and 61% were from "full blood" children. Hiller found the peak frequency for CXRs occurred in children aged one month to one year. Seventy-five percent of those X-rayed were less than three years old. Fifty-two percent of the films of the "full blood" children and 27% of the films of the "part Aboriginal children" were abnormal. Most of the abnormalities were due to pneumonia or atelectasis. Hiller made much of the point that acute bronchiolitis with over-inflated lungs was seldom found but he did not acknowledge that children with bronchiolitis may have normal X-rays (40). He described four children with non-resolving pulmonary collapse or definite chronic lung disease. Although 37% of the children had no follow up X-rays Hiller asserted that "there is very little chronic lung disease in the population". This was not a valid conclusion because the study reported incident disease and because the proportion lost to follow up was high. However the study did show that most paediatric pneumonia requiring hospitalisation in Aborigines occurred in children under three years of age.

In 1976 Hiller reported the CXR changes and radiological course for 19 of Maxwell's bronchiectasis patients following lobectomy (41). The progress of

the lobectomy patients was compared with that of 16 Aboriginal patients with bronchiectasis who were treated conservatively. The follow-up period was 2–8 years. Valid conclusions cannot be drawn from this paper because appropriate matching for severity of disease did not occur.

In 1976 Bateson *et al* (42) described the radiological findings in four Aborigines with bronchiectasis and total destruction of a lung. A second paper by Bateson in 1978 (43) reviewed 18 Aboriginal people with lobar or total lung destruction secondary to bronchiectasis. In nine of the bronchiectatic children (aged 2-10 at presentation) the mean interval from a previously normal CXR to unequivocal chronic lung disease was 2.7 years. Seven of the adult bronchiectatics (aged 28–60 at presentation) had a mean interval of five years from their last normal X-ray. Three of the adults and two of the children did not have specific respiratory symptoms at the time of diagnosis. Many presented with serious lung infection and cystic changes on their CXRs. Others had a history of pneumonia with incomplete resolution. Bateson *et al's* cases highlight one end of the spectrum of post-suppurative lung disease and, with Maxwell's series (6), provide the only published descriptions of bronchiectasis in Aborigines.

In 1976 Kamien reported a study of all the 0–15 year old Aboriginal children (n=410) living in Bourke, New South Wales (20). Each child's past medical history was ascertained from a semi-structured interview with the mother and perusal of their medical records in the Bourke District Hospital. The children were also examined for abnormalities of auscultation. Kamien found that 56% of 1–4 year old children had a history of "recurrent upper respiratory tract infection" (not defined), as did 21% of 5–9 year olds and 8% of 10–14 year olds. Adventitious sounds were heard in 32% of 0–4 year olds and 6.4% of 5–14 year olds. None of the children had a history of asthma. Although Kamien's findings differed from those of Gandevia (22), who found the prevalence of adventitious sounds remained high in 8–15 year olds, they demonstrated that the high prevalence of respiratory tract infections found in Papunya was not an isolated finding.

In 1976 Kamien also reported a study of the Aboriginal adults (aged 15 and over) in Bourke (44) . The total adult population was 320. The participation proportion in men was 91% and women 93%. Seventy-eight percent of the adults were current smokers. A history of chronic or recurrent respiratory disorder was obtained from 22% (58/264) of the adults. Specifically, 13% had a history of chronic bronchitis, 3% recurrent upper respiratory tract infections, 2% tuberculosis, 2% asthma, 2% recurrent pneumonia and 1 person had a

history of actinomycosis. The prevalence of loose cough averaged 20% and increased with age. Adventitious sounds were present in 17% and the prevalence also increased with age. In addition Kamien found that 24% of the adults had been admitted to Bourke District Hospital with respiratory disease at least once between 1967 and 1971. In itself this is an extraordinary finding. Kamien established that mass CXR screenings in Bourke of adults in 1968 and 1971 had found changes consistent with chronic bronchitis or emphysema in 6%, and tuberculosis in 2%.

Kamien's studies were the first complete epidemiological survey of respiratory disease in an entire Aboriginal Community and produced the first formal estimates of the prevalence of asthma and chronic lung disease in Aborigines. The radiological findings suggested at least 6% of adults had chronic lung disease. This was high as most studies of Caucasian adults reveal that the prevalence of chronic obstructive airways disease is 1-3% in women and 4-6% in men (45). Kamien's findings also suggested that the prevalence of asthma was approximately 0% in children and 2% in adults. This is low when contrasted with Peat *et al* who found the prevalence of current asthma in non-Aboriginal Australians to be 11.4% in children (38) and 6.3% in adults (46). None of the asthmatics Kamien identified were atopic. The author concludes from this study that the prevalence of asthma was low in Bourke and that the prevalence of chronic lung disease in adults was high.

In 1979 Chandler *et al* (26) investigated the respiratory function of 203 school age Aboriginal children living in Cherbourg Aboriginal community, 250 kilometres north-west of Brisbane. The study was designed to look for evidence of chronic lung disease (residual from childhood lower respiratory tract infections) and to develop models to predict FEV_1 and FVC. The study was restricted to 5–13 year old children attending school. Spirometry was performed using a Vitalograph dry spirometer. Only those with "consistent/satisfactory results" were recorded and an unknown number were rejected. Children with symptoms or signs of lung disease were not excluded. A group of 38 children had FEV_1 and FVC measured before and after bronchodilator. It was not reported how these children were selected. FEV_1 and FVC were presented as a percentage of the values published by Polgar and Promadhat (for Caucasian children). The FEV_1/FVC ratio was averaged for children by age group.

Chandler *et al* found that the FEV_1s in Cherbourg children ranged from 79 to 89% predicted. The FVCs ranged from 72 to 89% predicted. The mean

FEV_1/FVC ratio at all ages was greater than 97%. Three children had evidence of reversible airflow obstruction (not defined). Chandler *et al* concluded that widespread chronic airways disease and asthma were not present. Linear regression was used to model FEV_1 and FVC. The final models used height as the predictor. The R-squared values for the models and the standard error of the estimates were not reported.

In 1980 Chandler *et al* (47) reported the results of pulmonary function tests in 56 children from Mornington Island. Children were recruited from the schools as in the previous study and similar methods were used. Of the 92 children at school, 36 failed to perform satisfactory spirometry. The FEV_1s ranged from 75% to 89% predicted. The FVCs ranged from 76 to 87% predicted. The lowest mean FEV_1/FVC ratio was 92%. Chandler *et al* concluded that this study did not reveal any evidence of lung damage in children.

In the absence of any study reporting FEV_1 and FVC in healthy asymptomatic Aboriginal children Chandler *et al* had no accurate way of assessing the prevalence of abnormal lung function in the two studies described above (48). Therefore it cannot be concluded that Chandler *et al* found no evidence of chronic lung disease. In addition, the selection process for the two studies and the high rejection rate, may also have biased the findings towards healthier children. The prediction equations developed by Chandler *et al* (26) for FEV_1 and FVC cannot be assumed to predict "normal" lung function in Aboriginal children because they were not developed in children free of signs or symptoms of lung disease (48). However the studies do clearly suggest that FEV_1 and FVC in Aboriginal children are lower than corresponding predicted values for Caucasian children.

In 1983 Torzillo *et al* (23) studied 1,287 Aboriginal people living in the Pilbara, Western Australia. Most of the residents of seven communities participated. Subjects were examined for a loose cough, using the method of Gandevia (22), and auscultation of the lung bases was performed. Abnormal auscultation was defined as the presence of adventitae that did not clear with coughing. Lower respiratory tract abnormality was defined as the presence of loose cough and/or abnormal auscultatory findings (at two or more sites). The mean prevalence of loose cough was 17.6% (range 11.7-21.1), abnormal auscultation was 21.8% (range 10.0-30.2) and lower respiratory tract abnormality 28.5% (range 15.9-36.6). The prevalence of smoking in these communities was not reported. Torzillo *et al's* observations were similar to Gandevia's at Papunya. Both studies indicate widespread respiratory ill health. Without lung function

data it is not possible to fully assess the implications of these observations. As none of the children were reported to have rhonchi, the prevalence of asthma was probably low.

In 1984 Harris *et al* reported the incidence of childhood pneumonia for Aboriginal and non-Aboriginal children in Bourke (49). The records of 270 children (103 Aboriginal and 167 non-Aboriginal) born at the Bourke District Hospital in 1980 and 1981 were reviewed to record the number that had pneumonia in the first three years of life. Pneumonia was defined as an acute respiratory illness with symptoms, signs and radiological evidence of lobar or multilobar consolidation. Children with signs and symptoms of pneumonia but only "patchy" consolidation were excluded. Perinatal data and neonatal observations were also recorded.

Harris *et al* found that 25% of the Aboriginal children and 3% of the non-Aboriginal children had one or more episodes of pneumonia. Nine of the 26 Aboriginal children with pneumonia had experienced multiple episodes. The pneumonia in the Aboriginal children involved the right upper lobe in 77% of cases and other lobes were involved in 62%. The mean age of the first pneumonic episode was 14 months, and in more than half of the Aboriginal children the first episode occurred during the first year of life. Thirty-nine percent of the Aboriginal children with pneumonia weighed less than the tenth percentile for age. There was no correlation between birth weight, current weight, prematurity, breast feeding and pneumonia. The authors were not able to reach any conclusions regarding the impact of overcrowding and substandard housing because the numbers of children were small but the prevalence of pneumonia in children from substandard housing areas was relatively high (38%). This study clearly showed a differentially high cumulative incidence of pneumonia in Bourke Aboriginal children and was consistent with the anecdotal reports of similar problems in many Aboriginal communities (4).

In 1984 Clark *et al* (50) published an abstract which reported findings in 166 Aboriginal children (aged 7–16) from the Northern Territory. Clark *et al* performed histamine inhalation tests (for bronchial hyperreactivity) using the rapid method, and skin tests for atopy. The target population, sampling process, cumulative dose of histamine administered, criteria for BHR and the criteria for atopy were not reported. The allergens used were house dust, house dust mite, grass mix, cat, dog, and *Aspergillus*. The mean FEV_1 using (Caucasian based reference equations) was 84% predicted and the mean FVC

was 77%. Bronchial hyperresponsiveness was present in 16% (23/139) of children, and 42% were atopic. The prevalence of wheezing was not reported. The study suggested that prevalence atopy and bronchial hyperreactivity in Aboriginal children was similar to that in the non-Aboriginal population.

In 1984 Harris (51) reported examinations of nasal mucosa from 15 Aboriginal children with chronic lung disease. Samples of cilia were obtained by brushings from the nasal mucosa. All samples were examined immediately by direct microscopy and subsequently by electron microscopy. No immotile cilia were seen on the direct microscopy. In seven subjects minor abnormalities of cilial structure were seen by electron microscopy, and one subject had abnormalities consistent with the immotile cilia syndrome. Sampling details and the nature of the subject's chronic lung disease were not described and so the significance the findings is uncertain.

In 1986 Watson *et al* (52) reported lung function tests on 151 Aboriginal children aged 11-15 attending several schools in East Arnhem Land, in the Northern Territory. After a trial blow, the mean FEV_1 and FVC of the three subsequent blows was recorded using "the spirometer". The criteria for the acceptability of blows was not reported, nor if the results were in ATPS or BTPS. Children with symptoms or signs of acute or chronic lung disease were neither identified nor excluded. Data were also collected from 236 similarly aged non-Aboriginal children. Multiple regression was used to model FEV_1 and FVC in the Aboriginal children. It was assumed that the relationship between height and the logarithm of FEV_1 and FVC was linear. The R squared values for the equations were not reported, nor were the standard errors of the estimates. The FEV_1 values for the Aboriginal children were 15-17% less than those for the non-Aboriginal children. The FVC values in Aboriginal children were 17-22% lower than those of their non-Aboriginal counterparts. This study confirmed that spirometric values in Aboriginal children are probably lower than corresponding values in non-Aboriginal children. However these models (like Chandler *et al's*) cannot be assumed to define lung function in healthy Aboriginal children because children with signs or symptoms of chronic lung disease were not excluded.

In 1990 Harris *et al* (53) published a review of the changes that occurred in morbidity and mortality among the 0–14 year old Aboriginal children in Bourke between 1971 and 1984. Medical records were reviewed to establish the reason for hospital admission and to identify episodes of X-ray verified lobar pneumonia. In addition 100 households (representing 62% of Aboriginal

families resident in Bourke during 1984) were surveyed about their housing conditions.

Harris *et al* found that respiratory illness was the most common reason for hospital admission of 0-5 year old Aboriginal children in 1971 (847 per thousand) and 1984 (833 per thousand). For the non-Aboriginal children the admission rates for respiratory illness in 1971 were 91 per thousand and 1984 were 143 per thousand. Admissions for pneumonia in 0-5 year old Aboriginal children occurred with an annual incidence of 143 per thousand in 1968–1969 and 80 per thousand in 1983–1984. The most common organisms cultured from children with lower respiratory tract infections in Bourke were *Haemophilus influenzae* and *Streptococcus pneumoniae*.

Harris *et al* found that from 1971 to 1984 the proportion of 0–2 year old children with a weight below the tenth percentile fell from 43% to 29%, and in 3–9 year old children the proportion fell from 36% to 31%. A marked improvement in Aboriginal housing conditions also occurred from 1971 to 1984. The average number of persons per bedroom fell from 3.5 to 2.1, and the proportion with mains or septic sewerage increased from 59% to 75%. The proportion of houses with running water within five meters increased from 66% to 100%, and the proportion with a shower or bath increased from 60% to 90%. The availability of running hot water also increased from 50% to 84%. Access to electricity rose from 59% to 91% and refrigerator ownership rose from 27% to 93%. Harris *et al* hypothesised that the reduction in morbidity seen in Bourke Aboriginal children from 1971 to 1984 was related to improvements in housing conditions, increased access to running water (and other amenities) and improved nutrition. These findings are important because this is the only evidence to date that improved respiratory health in Aboriginal children might be associated with improved living conditions.

In 1992 Thompson *et al* (1) measured ventilatory function in 70% of the adult population of an isolated Queensland Aboriginal community. A total of 288 adults aged 20 and over had FEV_1 and FVC measured using a Vitalograph wedge spirometer. The aim of the study was to model ventilatory function in healthy adults. FEV_1 and FVC were measured according to the ATS recommendations and all results were reported in BTPS. Subjects with known lung disease, loose cough, chronic cough, or adventitae on auscultation, or FEV_1/FVC ratio less than 60%, were excluded. Following the application of these exclusion criteria, 229 subjects were available for modelling. Eighty-one percent were smokers. Thompson *et al* found that the FEV_1 and FVC in the

Aboriginal adults were approximately 70% of that predicted by Caucasian equations. Thompson *et al* found that FEV_1 declined with age in smokers and non-smokers but that the decline was apparently greater in non-smokers. The effect was sufficiently marked that by the age of 35 years smokers had better lung function than non-smokers. The authors were surprised by this finding because cigarette smoking is not generally believed to retard the decay of spirometric function.

2.5 Summary

2.5.1 Pneumonia

In Aboriginal children there is good evidence that the incidence of pneumonia is high in comparison with non-Aboriginal Australian children (24, 35, 39, 51, 53). Although pneumonia is an important cause of infant mortality in Aborigines it is more frequently a cause of premature mortality in adults (28). It has been shown that most Aboriginal childhood pneumonia occurs in the first three years of life (35, 39, 49, 53). Although there is some evidence to suggest that pneumonia in Aboriginal children is associated with substandard housing (49, 53) little is known about the cause(s) of the high attack rates. No adequate studies have been performed to determine if early childhood pneumonia in Aborigines is associated with abnormalities of lung function that persist into adulthood.

In Aboriginal adults pneumonia is an important cause of premature mortality and hospitalisation (24, 27, 33). It is not known why the incidence of pneumonia is high or if these infections are a cause or a result of chronic lung disease. Chronic lung disease may be an important risk factor for pneumonia in Aboriginal adults.

2.5.2 Bronchiectasis

There is 25 year old evidence from one community to suggest that the prevalence of bronchiectasis in Aboriginal children may have been as high as 2% (35). It has been hypothesised that bronchiectasis in these children is due to lower respiratory tract infection and concurrent dehydration (6). There is no evidence that bronchiectasis in Aboriginal children is caused by ciliary defects, hypogammaglobulinaemia or cystic fibrosis (6, 51). The sensitivity and specificity of crepitations in Aborigines for detecting bronchiectasis or other chronic lung diseases is unknown. The current prevalence of bronchiectasis in Aboriginal adults and children is also unknown as is the role of bronchiectasis in the aetiology of pneumonia.

2.5.3 Asthma

Evidence from clinical studies suggests that the prevalence of asthma in Aboriginal children is less than 4% (20-23, 26, 35, 47). This means it is significantly lower than that found in non-Aboriginal children (see chapter 6). This is surprising because the only study of BHR and atopy in Aboriginal children suggests that the prevalence of these abnormalities (commonly associated with asthma) is very similar to that found in non-Aboriginal children (50). In adults, the only recorded estimate for the prevalence of asthma is 1.9% in Bourke by Kamien (44). Thus the prevalence of asthma in adults and children appears low when contrasted with that in non-Aboriginal Australians. There is a need for further research to confirm these findings and to explore the relationship between asthma, atopy and BHR in Aborigines.

2.5.4 Normal Lung Function

The three studies of ventilatory function in Aboriginal children all suggest that FEV_1 values are approximately 11-25% lower than those found in non-Aboriginal children and that FVC values are 11-28 percent lower (26, 47, 52). Although lung function was modelled in two of these studies (26, 52) the equations cannot be assumed to accurately predicted normal lung function because children with symptoms or signs of lung disease were not excluded.

There is only one study of ventilatory function in clinically well Aboriginal adults (1). That study suggested FEV_1 and FVC in Aboriginal adults are 30% lower than corresponding values for Caucasians and that spirometric function declined faster in non-smokers than smokers. Further research is required to confirm these findings and to explore the impact of cigarette smoking on spirometric function in Aborigines.

2.5.5 Chronic Lung Disease

Plant (27) has shown that Aboriginal males in the NT are 5 times and females 11 times more likely than their non-Aboriginal counterparts to die from COPD and asthma. Devaneson *et al* (24) showed that pneumonia, COPD and asthma were the main respiratory reasons for Aboriginal hospitalisation in the NT. It is likely that COPD and asthma are the most prevalent chronic lung diseases in NT Aborigines. The CXR component of Kamien's survey in Bourke (44) suggested that the prevalence of chronic lung disease in adults there was at least 6%. Also Maxwell's 1968 CXR survey (35) of children in Papunya suggested the prevalence of chronic lung disease in children could be at least 6%. The nature and significance of the diseases identified in these CXR surveys is not known. The work of Maxwell and Kamien suggested that some of the

people with abnormal CXRs had bronchiectasis and some had COPD. Because clinical experience shows that CXRs have poor sensitivity for detecting people with mild ventilatory dysfunction it is likely that the prevalence of chronic lung disease(s) was even higher than that suggested by these surveys.

2.6 Conclusion

There is a large body of evidence to suggest that respiratory diseases are an important cause of morbidity and mortality in Aboriginal Australians. However almost nothing is known about the causes, nature and distribution of lung diseases within the Aboriginal population.

CHAPTER 3

Communities, Consultation and Feedback

3.1 Introduction

This chapter begins with a review of the important ethical issues in Aboriginal health research. The next section contains an overview of the participating communities and the rationale for their selection. The chapter concludes with a description of the process utilised to obtain informed consent and return results.

3.2 Ethics

Recently developed guidelines for health research in Aboriginal communities (3) emphasise the importance of consultation, community involvement, informed consent and providing feedback. The guidelines also stress the importance of ensuring that tangible community benefits, such as employment for local people and health promotion, accompany research. The present study was conducted in accordance with these guidelines.

The guidelines cited above were developed following a large Aboriginal health conference in 1986 in Alice Springs, NT. At that meeting Aboriginal people expressed distress and hostility about research activities in their communities. Strong resentment was expressed because Aboriginal people were not being consulted about research. Examples were given of researchers proceeding with studies against the explicit instructions of community elders. It was also observed that once researchers had collected "their" data, Aboriginal people

rarely heard from the researcher again. Those performing research for the degree of Doctor of Philosophy were particularly singled out for criticism. It was against this background that formal guidelines for research in Aboriginal communities were developed.

This project was approved and financially supported by the Australian Institute of Aboriginal and Torres Strait Islander Studies. The research was also approved by the Aboriginal Coordinating Council in Queensland and the appropriate community councils. In addition approval was given by the Australian National University ethics in human experimentation committee.

3.3 Background & Rationale for Community Selection

The diversity in living conditions and the small size of many Aboriginal communities pose problems for studying the epidemiology of chronic disease. The pooling of data from several communities potentially reduces these difficulties. To maximise numbers and to examine regional variation in the prevalence of abnormalities, the author selected four contrasting communities for the present study. For confidentiality the communities are referred to as Cape York 1 (CY1), Cape York 2 (CY2), central Australia 3 (CA3) and central Australia 4 (CA4).

CY1 and CY2 were selected because a colleague, Dr Thompson observed that asthma was a significant health problem in CY1 but not in CY2. Puzzled by this observation and after discussing the issue with community representatives, he sought support to investigate asthma and other lung diseases in these communities.

CA3 was approached because the members of this "traditional" community have been reported to be in relatively good health (32). It was also approached because the author had visited the community during the 1980's and personally knew several colleagues who had worked there for extended periods.

CA4 was selected to assess the impact of particularly adverse living conditions and petrol sniffing on respiratory health. It was also a logical choice because the author and his wife were medical officers in its community controlled health service during 1986 and 1987.

The following section contains a précis of life in each of the participating communities. Most of the information was obtained during discussions with Aboriginal community informants. A consideration of these varying circumstances led the author to deem that living conditions in CY1 were

superior to those in the other three communities and that CA3 was better than CY2 and CA4. The author hypothesised that the prevalence of abnormal lung function (ALF) would reflect this by being relatively low in CY1 and CA3 and high in CY2 and CA4.

3.3.1 Cape York 1 (CY1)

CY1 is located in a rainforest on the eastern Queensland coast approximately 60 kilometres from Cooktown. The climate in CY1 is tropical with a high rainfall, a long humid wet season and mild winters. At the time of the study the community had a population of approximately 700 people. The community comprised many Aboriginal tribes living on the traditional lands of the Guugu Yimidhirr. Its residents had good literacy and numeracy skills and English was the main language.

The tribes from around CY1 were some of the first Aborigines to encounter Captain Cook in 1770. The traditional people of the region were decimated during the Palmer River gold rush of the 1870s. In 1881 the land in the area was gazetted as an Aboriginal reserve to create a refuge and in 1885 the first missionary arrived. Since that time the residents of CY1 have been coerced into adopting a European lifestyle. During the second World War all the residents were temporarily relocated further south. The community ceased to be a mission during the 1980s. At the time of the study the residents of CY1 had no legal tenure over their land.

At the time of the present study most people from CY1 lived within the community in well maintained houses with functioning showers and toilets (see table 3.1 and illustration 3.1). Overcrowding was not prevalent. Generally the houses had three bedrooms with uncarpeted wood or concrete floors. Most people slept inside on mattresses placed directly on the floor. Electricity and gas were the principal energy sources for cooking and heating was not required.

Despite the generally good standard, it was apparent that there was a range of living conditions in the community. At one end of the spectrum were people who were more westernised, lived relatively well and ate every day. In contrast was a socially depressed group who frequently drank alcohol at week-

Illustration 3.1 The upper photograph shows the main street in CY1 and the lower one depicts an average house.

ends and often went without food. Alcohol was not available in the community but was obtainable within an hour's drive. Alcohol abuse was not generally considered to pose a threat to the maintenance of law and order or community cohesion.

Although most food came from the community store, bush foods were an important source of weekend nutrition. Many people went fishing or hunted for kangaroo and pigs. During the week people generally ate bread and jam accompanied by tea and sugar for breakfast. For the main meal people generally ate hot stew, fish, chicken, or fried steak, with rice or fried chips or damper with jam. Take-away prepared meals such as pizzas and pies were also popular.

Employment opportunities in the community were limited and primarily confined to clerical tasks in the community office or maintenance of community facilities.

CY1 had a small hospital that was staffed and run by the Queensland Department of Health. It employed local Aboriginal health workers but was not under community control. A doctor from Cooktown visited twice a week. Staff from the hospital rarely saw pneumonia in children. Asthma in adults was a commonly recognised problem.

3.3.2 Cape York 2 (CY2)

CY2 is located on the coast of the Gulf of Carpentaria at a similar latitude to CY1. Like CY1 the climate is tropical. At the time of the study the community had a population of approximately 400 people. The main languages were Tior and Munkin and compared with CY1 the people had relatively poor English literacy and numeracy skills.

Significant contact with Europeans occurred later than for CY1 and coincided with the arrival of pastoralists in the 1880s. In 1897 the coastal lands of the Gulf were set aside as Aboriginal reserves and in 1938 a Church of England mission was established at the site that is presently CY2. By 1956 the majority of the indigenous population were living in simple palm thatched shelters within the mission. In 1964 a cyclone devastated the mission destroying most of the dwellings. The Queensland Government promptly rebuilt a community store and erected three bedroom prefabricated aluminium houses for most of the residents.

Illustration 3.2 The upper photograph depicts above average housing in CY2 and the lower one shows inadequate housing in the same community.

The community remained a mission until 1967. At the time of the study the Tior and Munkin people did not have legal tenure of their land. The residents of CY2 lived in 87 houses and many of them were in poor condition. Less than 50 per cent of the houses had functioning showers, but most had toilets (see table 3.1 and illustration 3.2). Most of the houses comprised three bedrooms and people slept inside on mattresses on the floor. The floors were made of concrete or wood and not carpeted. Electricity and gas were the principal modes of cooking and heating was not required.

Since 1960 most food consumed by residents has been purchased from the community store but at the time of the present study bush foods remained an important source of food on weekends. Hunting for pigs and wallaby was popular sport and supplemented food supplies when money was scarce. For breakfast people generally ate bread, damper and jam or cereal with sugared tea. For the main meal people ate tinned meat and rice or pies and chips. At the time of the study alcohol was freely available within the community on three days each week. Alcohol abuse was considered a serious problem because it disrupted community cohesion and law and order.

Employment opportunities in the community were limited and most jobs were low skilled. These included the maintenance of essential community facilities or community services such as the school, council office, store, women's resource centre and police. A near by crocodile farm, the community hospital and the canteen (pub) provided other sources of employment.

The community hospital was run by professional staff from the Queensland Department of Health. It employed local health workers but was not under community control. Staff from the hospital reported that pneumonia in children was uncommon but that recurrent chest infections and bronchiectasis in adults was prevalent. Asthma was not known to be present among the indigenous people of this community.

3.3.3 Central Australia 3 (CA3)

CA3 is located 350 kilometres from Alice Springs in the NT. The climate is arid with subzero temperatures in winter. At the time of the study it had a population of approximately 800 people living in 14 small communities called outstations. These outstations were scattered over 1800 square kilometres of desert. Outstations comprised a group of houses and traditional shelters (called warles) where family groups and friends lived close to areas of

traditional significance. CA3 has never been a mission. The main languages spoken were Alyawarra and Anmatyerre and most people had only limited English literacy and numeracy.

The lands of CA3 were expropriated to create a pastoral lease in 1927. The traditional owners of the land remained and lived in strained coexistence with the pastoralists until 1979 when the community gained permanent legal title to the leasehold.

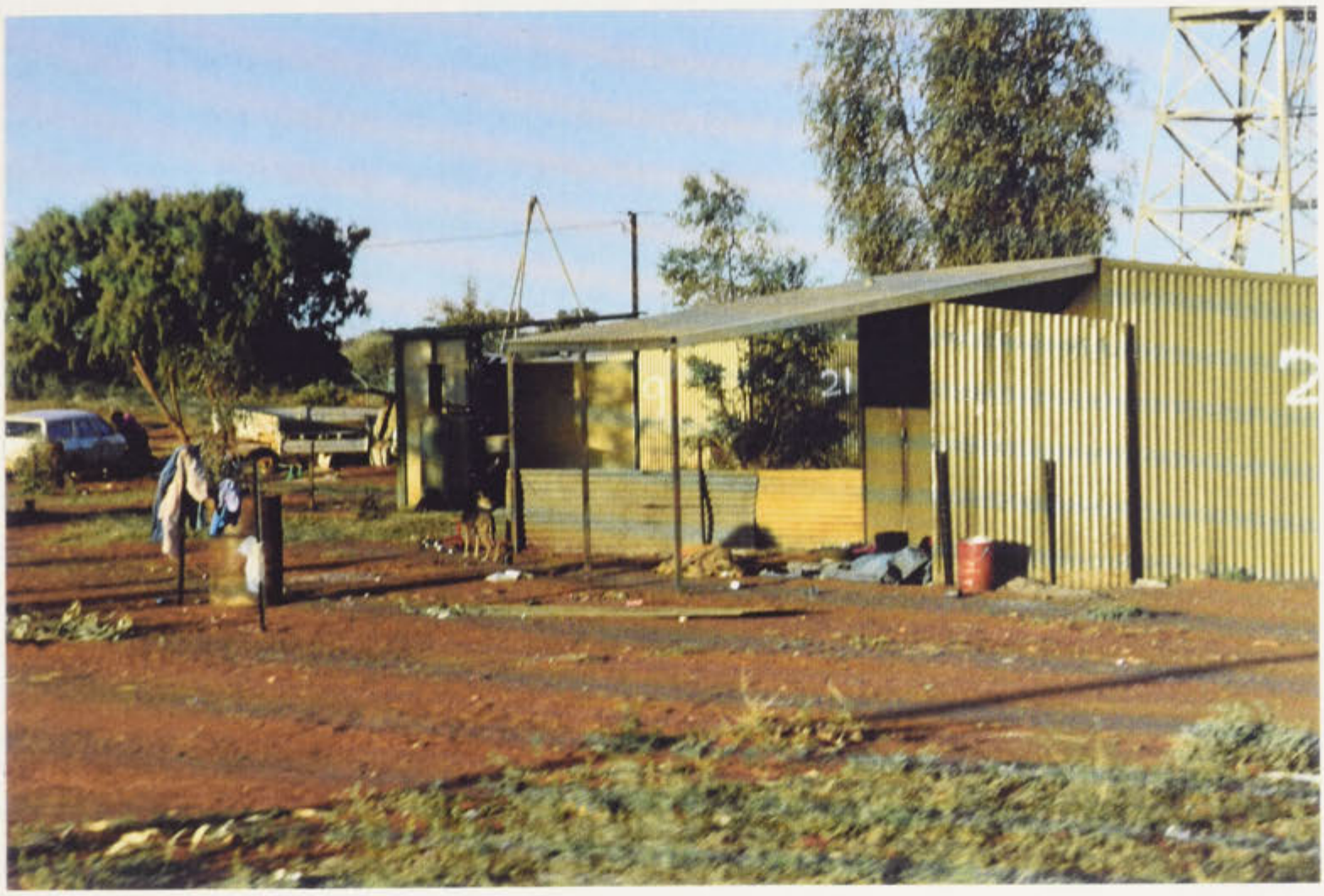
The community houses were built on concrete slabs, had tin or brick walls and wide verandas (see illustration 3.3). Generally the houses had no floor coverings and minimal furniture. Most people slept on mattresses on the floor. Fire was both the principal source of heating and method of cooking. The author was unable to determine the number of houses with functioning showers and toilets.

Bush foods have been shown to comprise approximately 40% of the diet in at least one of the outstations in this community (32). At the time of the present study native vegetables were collected when available and hunting for kangaroo was a regular activity. The remainder of the food came from the community store. The community has a long standing policy of ensuring healthy foods are available in the store. At the time of the study alcohol was not available throughout CA3 and alcohol abuse was not widespread. However increasingly, young men were engaging in episodic heavy drinking.

There were very few employment opportunities in this community. However the community does have a strong tradition of painting and revenue from this and other artistic endeavours is valued.

The community health service was run by the NT Department of Health and employed a doctor, several sisters and Aboriginal health workers. Staff from the health service reported that pneumonia in children under the age of two was common but rarely severe enough to warrant evacuation to Alice Springs. Asthma was recognised in adults but was unknown in children.

Illustration 3.3 The upper photograph shows typical housing in CA3. The lower photograph is an example of the type of housing that was gradually being introduced to the community at the time of the present study.



3.3.4 Central Australia 4 (CA4)

CA4 is located 400 kilometres from Alice Springs on the Anangu Pitjantjatjara Lands in South Australia. The community lies in one of the most arid regions in Australia. The annual rainfall is less than 150 mm and the summers are long and hot (the mean temperature in January is 37 degrees Celsius). The winter temperatures fall below zero. At the time of the study CA4 had a population of approximately 350 people. The main languages spoken were Pitjantjatjara and Yankunytjatjara. The majority of the residents had poor English literacy and numeracy skills.

Regular contact with Europeans began in the region of CA4 after the completion of the overland telegraph in 1872. In the early 1920s "inviolable reserves" were gazetted because of concern that European contact would "degrade" the traditional owners of the land. In 1937 a mission was created approximately 100 kilometres from the present site of CA4. After World War Two government intervention sought to end the nomadic existence of those living in the reserve. CA4 was one of many mission communities created on the reserve in the 1960s to "train the people to become useful citizens" (54). Since the 1970s there has been some government recognition of the adverse effects of forced concentration of Aboriginal people and some support for the decentralisation into smaller family units on "homelands". In 1981 the Anangu Pitjantjatjara won freehold title to their land.

At the time of the present study approximately a third of the population lived on homelands within 50 kilometres of the central community. The residents of CA4 generally lived in inadequate poorly maintained houses or in traditional shelters called wiltjas. The quality of housing in the community varied from good to derelict (see table 3.1 and illustration 3.4). Most of the houses had two bedrooms but the majority of people preferred living and sleeping outside the house, when the weather permitted. The houses had wooden or concrete floors without coverings. Fire was the principal means of heating and cooking. At the time of the study only 31% of the houses in CA4 had functioning showers and toilets.

At the time of the study bush foods made a small but significant contribution to the daily diet in CA4. Honey ants and native vegetables were collected when in season and witchetty grubs (maku) were a sought after delicacy. Hunting for kangaroo (malu) was a regular activity because kangaroo meat is an import-

Illustration 3.4 These photographs show typical housing in CA4.

ant part of the local diet. Most processed food was purchased from the community store although food was also available an hour's drive from the community. For breakfast people generally ate cereal or porridge with sugar. Mid-morning people had grilled or stewed meat with bread or damper and jam with sweetened tea. Another meal was eaten before dusk if food was plentiful. Alcohol was not freely available in the community but it was obtainable within an hour's drive. Alcohol abuse has been increasing since 1986. The inhalation of petrol fumes (known as petrol sniffing) was a prevalent form of substance abuse among adolescents at the time of the study. Petrol sniffing has been occurring in the community for at least twenty years.

Employment opportunities in this community were also limited.

At the time of the study the community had its own health service which employed three sisters and six community health workers. CA4 was the only community to have a community controlled health service. The health staff in CA4 observed that nearly all children under the age of two years experienced at least one episode of pneumonia. Each year several cases were severe enough to warrant evacuation to Alice Springs Hospital. Some adults had asthma but childhood asthma was not a recognised problem.

Table 3.1 Regional variation in the provision of basic facilities.

	CY1	CY2	CA3	CA4
Population	700	400	800	350
Number of houses	111	87	*	26
Number of wiltjas/warles	0	3-6	*	12
% carpet	~0%	~0%	*	~0%
% houses with water	100%	100%	*	42%
% with hot water	85%	55%	*	21%
% with functioning showers	100%	100%	*	31%
% with toilet	100%	100%	*	31%
% with washing machines	75%	33%	*	<10%
% with fridges	75%	63%	*	<10%

* unknown

3.4 Informed Consent

Each community was initially approached informally to explore their interest in a lung health study. The author then wrote directly to the appropriate community councils outlining the aims and methods of the proposed study. Permission was obtained to visit each community for a week to explain the proposal and to discuss the most appropriate way of obtaining informed community consent.

3.4.1 CY1

Initially Dr Thompson approached elders in CY1 and CY2 to discuss the possibility of performing a respiratory health study. These discussions confirmed that general respiratory health issues (particularly asthma) were of interest to community members. Having received "in principle" support for a study, Dr Thompson wrote to the Aboriginal Coordinating Council (ACC) in Cairns seeking permission to proceed with further consultation.

In March of 1990, after receiving permission from the communities, the author arranged to visit CY1 and CY2 for a week in May. The aim of the visit was to gauge community support for the proposed study and to assess the feasibility of the proposal. It was also an opportunity to arrange employment of local Aboriginal staff. The author developed and piloted the initial draft of the questionnaire during this preliminary visit.

On arrival in CY1 the author first made contact with the health service. Following a suggestion made by the clinic staff he took an instamatic camera and art materials with him. The health workers photographed the author and themselves demonstrating each step in the proposed study protocol. The pictures were pasted onto brightly coloured cardboard and annotated by the health workers who displayed them around the community. The posters proved popular and created a focus for community interest and comment.

The author was next introduced to the important community members by Aboriginal staff from the health service. An early meeting with the community chairman facilitated the consultation process. The community chairman suggested that the author discuss the proposal with the headmaster of the school and arrange for consents to be sent home with children in the week prior to the study. He also suggested that provision be made for adults to sign consent forms on the day of testing and that the author make a video about the proposed study. This was subsequently done at the Australian National University's imaging resources unit. The community chairman then arranged

for the video to be broadcast before the children's consent forms were sent home, and again during the week prior to data collection. The video was available for viewing in the community hospital and was replayed several times during the data collection period.

By the end of the initial visit the author was confident that the study proposal was understood and acceptable. Most people appeared to appreciate that asthma and lung disease were worthy of investigation because almost everyone knew somebody with respiratory symptoms. In this community many people told the author that asthma was caused by allergy to mango tree flowers and bush turkey dust.

3.4.2 CY2

The author also allocated a week for consultation in CY2 and Dr Kingsley Whittenbury, a general practitioner with experience in Aboriginal Health, accompanied him on the visit.

As in CY1, the indigenous community health workers and other clinic staff played an important role in introducing the author to the community. Again, posters were made which included photographs of health workers simulating each step of the proposed study. The activity proved to be a relaxing way to initiate relationships with the health workers.

The community adviser and chairman were both supportive and made many practical suggestions. The council also asked the author to produce a short video to explain the study. After speaking with the school's headmaster the author saw each class and discussed lung disease, smoking and the proposed study. Also the author was interviewed about the proposal on the community radio station.

At the end of the week the author was convinced that the community was interested in the proposed study. However in CY2 it was clear that respiratory health issues were not viewed with as much importance as they were in CY1.

3.4.3 CA3

After informal discussions with two colleagues who had worked in the community, the author wrote to the administrator of the health service with a detailed outline of the proposed study. After obtaining "in principle" support the author arranged a visit to discuss the project in July 1991.

As CA3 is decentralised the author travelled to the various outstations with the health service staff on their routine visits. This facilitated introduction to key people in each of the camps. The author employed two people (one current and one former health worker) to interpret and facilitate the discussions.

Generally the community members were enthusiastic about the study and showed a keen awareness of the importance of a high participation rate. (Several people said, "it is very important that you see everybody in each camp otherwise you may miss something.") The author received many helpful suggestions for overcoming practical problems posed by the lack of reticulated power and the rudimentary facilities. For example in some outstations we were invited to do the testing in the school (which had generators) and in others disused sheds or the shelters used by the health workers were made available.

It was impossible to achieve consensus on the importance of written consent in this community as people felt it was unnecessary since "consent" was implied by participation. In deference to the Aboriginal and Torres Strait Islander Health Research Guidelines the author offered written consent forms to all participants at the time of enrolment but did not insist that the form was signed.

3.4.4 CA4

In CA4 the consultation process was facilitated by the author's familiarity with the community. In 1990 the author wrote to the director of the CA4 health service, the CA4 community council and to the regional office of the Nganampa Health Council Inc. (NHC) in Alice Springs. The research proposal was circulated to the Menzies School of Health Research and the Central Australian Aboriginal Congress (Congress medical service) in Alice Springs. Although the response to the proposal was generally favourable, Congress medical service expressed the view that the proposal contained little of immediate benefit to Aboriginal people. Finally the proposal was approved at a regional NHC meeting and permission was granted for the author to proceed with the consultation process.

In May 1991 the author visited the community for a week to discuss the proposal. The study was discussed at an open community meeting. This was the only community, of the four studied, that regularly held such meetings. The issue of informed consent was debated at length and it was agreed that a collective consent was appropriate because literacy skills were poor. It was

clear that individuals retained the right to decline. The author also visited the CA4 outstations to discuss the proposal with those who had not been able to attend the community meeting.

3.4.5 Summary

This research proposal was received favourably and finally accepted because:

- The author was known to each of the communities or their networks;
- The author was seen as credible having worked in an Aboriginal community;
- Each community was given an opportunity to discuss and influence the research design;
- The proposal was presented to the communities via community councils and health services;
- Sufficient time was allowed for the consultation process;
- Respiratory health issues were of interest to each of the communities;
- Aboriginal people were employed throughout the consultations and plans to include Aboriginal health staff in data collection were made explicit;
- The proposal included provision for appropriate individual and collective feedback.

3.5 Feedback

The obligation to provide collective and individual feedback was addressed early because all communities saw this as an important objective. Initially each subject received a verbal summary of their respiratory health status at the time of the data was collected. In addition the health consequences of smoking were discussed with each participant (with an interpreter present) following the measurement of tidal carbon monoxide levels. Arrangements were made for referral and treatment of any urgent health problems revealed by the testing. Health staff in each community were informed about all subjects identified with chronic lung disease or asthma.

Some months after the data collection each community was visited for a week to provide further feedback. Written reports were produced for each participant. The reports contained a technical section and a simple interpretive summary. A copy was made available to each participant and a second copy was inserted into their clinic or hospital record. These reports contained a summary of the participant's findings. The author was available for individuals to discuss their results. Videos summarising the important health consequences of the research were made for CY1 and CY2 (as requested) and these were broadcast over the community television stations and retained for use as teaching aids in the clinics and schools.

3.5.1 CY1

In CY1 it was suggested that the author set up a "feedback station" in the Aboriginal Health Program office. During the week 180 people (33% of the participants) returned for their reports. This opportunity was used to explain the report and to reinforce the earlier discussion about the health effects of smoking with smokers. At least two people had given up smoking in the interval between the data collection and feedback visit.

During the feedback visit the author spoke to members of the council, health workers and the sisters about the results of the study. He was invited to talk to the school children about respiratory health and disease. All classes received two lessons about basic lung structure and function, asthma and the health effects of smoking. Copies of a large diagram of the lung were used as a teaching aid and the children were given a chance to listen to each other's chests.

3.5.2 CY2

In CY2 the author was advised to post the reports through the community mail service and display an explanatory poster in the community office. People were invited to the clinic during the week to discuss their own findings but few took this option. The other copy of the report was incorporated into the case notes. As in CY1, the author was encouraged to talk in the school, the clinic and to community elders. The hospital staff were very interested in the results and welcomed the video for teaching.

3.5.3 CA3

In CA3 the feedback process was complicated as the medical and nursing staff had completely changed since the time of the data collection. The new clinic staff welcomed the author's visit and found the respiratory health overview was helpful. As in CY2, most people showed little interest in receiving or discussing their written report but many wanted to be reassured that a copy was in their medical record. Many people took delight in recalling their tidal carbon monoxide level. Community elders were pleased to hear that the community had relatively good respiratory health by the present study standards. They interpreted this as vindicating their lifestyle.

3.5.4 CA4

In CA4 people were interested in the collective results and implications more than their individual results. Many people thought that the results should be used to challenge the community council and support agencies to improve living their conditions. The author was asked to write a letter to the council summarising the study's findings and the deficiencies in the community infrastructure.

3.5.5 Summary

- Immediate feedback at the time of data collection was appreciated;
- Permanent reports for medical records were valued because they were seen as a resource for health service staff. In communities where literacy levels were low written reports for personal use were not valued;
- Informal verbal feedback was the most appropriate feedback for many people;
- With time interest in these data shifted from a focus on individual findings to a consideration of the collective implications;
- Community elders placed a high priority on the education of children about health issues raised by the study;
- Health service staff found the research process stimulating because they valued the insights it brought to health problems in the community;
- The potential for research to be used to lobby for lifestyle support or change was recognised;
- The provision of feedback is a necessary, rewarding and valued part of the research process but it is time consuming and requires planning and consultation to be appropriate.

CHAPTER 4

Sampling and Participation

4.1 Introduction

Defining "the population" in Aboriginal communities is difficult because Aboriginal people maintain their traditional mobility (55). Although most communities have population registers, experience shows that they tend to over-estimate the size because people are often registered in more than one community. In the present study the local health service estimates of the number of people "usually" living in each community were used as the population denominator. Although these estimates were undoubtedly imprecise they were the best available.

4.2 Sampling

In the present study every Aboriginal person aged five years and over living in CY1, CY2 and CA4 was eligible to participate. The author anticipated this would minimise sampling bias and maximise the power of the study.

In CA3 it was not feasible (because of the decentralisation and rudimentary conditions for data collection) to study the entire community in the available time. Following discussions with community representatives a pragmatic decision was made to study half the outstations. During his preliminary visit the author accompanied CA3 health staff on their routine clinics and invited the residents of the first eight outstations to participate. Seven outstations accepted and one gave qualified approval because most of the residents were away at a land council meeting. In the selected outstations all people aged five years and over were deemed eligible for participation. During discussions held at the

commencement of the data collection visit (some months later) community informants reported that the outstation which had earlier given tentative approval no longer wished to participate*. This outstation was therefore excluded from the sampling frame. Although the selection process was not random, those studied were likely to be representative because community informants regarded the outstations of CA3 to be very similar.

In CY1 and CY2 a week was allowed for data collection in each community. In CA3 and CA4 two weeks were allocated because the communities were more decentralised than those in Cape York and because insufficient time in CY2 contributed to a disappointing participation rate there (see below).

4.3 Participation

4.3.1 CY1

In CY1 the community response was positive and 80% (478/600) of those eligible were tested (see table 4.1). The age range of the sample was 5-80 years and the male to female ratio was 0.92. The age and gender structure of the sample suggested that 15-34 year old males and 15-19 year old females were under-represented (see figure 4.1). Several people with known lung disease did not participate because they believed that they had little to gain from further investigation since they had already seen Dr Thompson (who was part of the data collection team in CY1 and CY2) on past visits. Many of the non-responders were not present in the community and a few told the health workers that they did not wish to participate.

4.3.2 CY2

The community response in CY2 was less enthusiastic than that in CY1. After a week of data collection 51% (163/320) of those aged five and over were tested (see table 4.1). The age range was 5-60 years and the male to female ratio was 0.94. The age and gender structure of the sample suggested that 5-9 year old males, 15-19 year old males and females, and 25-29 year old males were under-represented (see figure 4.2).

Numerous factors contributed to the low participation rate in CY2 but unexpected competition from other community activities was the principal problem. When the field team arrived on the Sunday prior to the appointed

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* It was not made explicit why this community chose to withdraw but it was probably because they didn't feel they knew enough about the proposal.

week for data collection we were disappointed to find that half the population was attending a football match in a second community two hour's drive away. No-one was certain when everybody would be back. We also learnt later that day that the visiting magistrate had also arrived and would be sitting for three days to hear charges against community members. To make matters worse the pivotal elder who had agreed to assist us was required in court because he was a Justice of the Peace. Thus for the first three days of the week the community was preoccupied with returning from football and attending the hearings. The final challenge to the study came from a religious convention that commenced on the Wednesday just as the court was recessing. It was Friday afternoon before there were no other competing activities in the community.

4.3.3 CA3

The community response in CA3 was supportive and 72% (358/500) of those aged five years and over were tested by the end of two weeks (see table 4.1). The age range of those tested was 5-84 years and the male to female ratio was 0.98. The age and gender structure of the sample suggested a deficiency of males and females aged 20-24 years and males aged between 25-29 and 40-44 years of age (see figure 4.3).

In one outstation the chairman sanctioned the commencement of data collection one afternoon but withdrew the permission on our return the following morning. He told the author that the residents were anxious about what the tests might reveal (eg cancer), so testing was abandoned in that outstation. In all the other outstations the response was positive with participation rates of approximately 90% percent. When the residents of the "withdrawing" outstation were included in the sample the participation rate for CA3 fell to 72%.

4.3.4 CA4

In CA4 the community response to the study was very positive. After two weeks of data collection 87% (260/280) of all people aged five years and over had participated (see table 4.1). The age range of the sample was 5-76 years and the male to female ratio was 0.92 (see table 4.1). The age and gender structure of the sample revealed a minor deficiency of children aged 10-14 years and 20-34 year old males. Of those who were not tested some were away from the community and some declined without explanation.

4.4 Discussion

Each community sample was divided into 5 yearly intervals for comparison with the 1986 census for Northern Territory Aborigines (see table 4.2). This comparison confirmed the impressions regarding the deficiencies of the samples gained from examination of the population trees for each community (see above). That is, that under-representation occurred in CY1 and CY2 among 15-19 year olds and in CA3 among 20-24 year olds. Otherwise the age structure of the samples reflected that of the wider Northern Territory Aboriginal population.

In general non-participation was due to absence from the community, anxiety should disease be revealed or commitments to other activities of higher priority. In CY1 a few people who were known to have lung disease did not present for testing as they had seen Dr Thompson on previous visits. However the participation rates of those with known lung disease in CY1 matched the participation of those without known lung disease. Therefore it was unlikely that non-participation introduced significant bias in CY1.

The generally high participation rates in this study partly reflected the time devoted to consultation and planning, the perceived importance of respiratory disease, the involvement of community members in the field team, and the promise of individual and collective community feedback.

The relatively poor participation rate in CY2 was mainly due to the influence of other competing activities although it also probably reflected the author's early impression that respiratory health issues were not regarded with the same level of interest as the other communities. In retrospect allowing more time to offset the multiple activities competing for peoples' attention would have improved participation rates. The poor participation rate in CY2 means that one has to be cautious about interpreting the findings from this community. The potential effect of sampling bias on the results from CY2 are discussed in chapters 5-10 where relevant.

4.5 Summary

Data were collected in four rural Aboriginal communities from 1,252 individuals with an age range from 5 to 84 years and a male to female ratio of 0.93. The proportion of 5-84 year olds participating in the four communities ranged from 51% to 87%. The age breakdown of the sample from each community was similar to the Aboriginal population of the Northern Territory. However, 15-19 year olds (especially males) are under-represented in CY1 and

CY2, and 20-24 year olds (especially males) were under-represented in CA3. The sampling problems encountered in the present study highlight the importance of adequate consultation in Aboriginal health research and the importance of allocating sufficient time for data collection. The precise effects of non-responses in the present study are unknown. The magnitude of the non-response in CY2 dictates caution with interpretation of the findings from this community.

Table 4.1

Participation by community.

Participation Data	Community			
	CY1	CY2	CA3	CA4
Date of testing	8/90	8/90	8/91	9/91
Population¥	600	320	500*	290
Males tested	229	80	177	119
Females tested	249	83	181	134
Total tested	478	163	358	253
Participation	80%	51%	72%*	87%
Age range	5-80	5-60	5-84	5-76
M:F ratio	0.92	0.94	0.98	0.92

* selected outstations

¥ estimated population aged five years and over.

Table 4.2

Comparison of the age structure of each community sample with that of the Northern Territory Aboriginal Population from the 1986 Census.

	CY1	CY2	CA3	CA4	ABS
Ages:					
5-9	19	17	14	15	14
10-14	16	15	17	11	14
15-19	8	7	17	15	13
20-24	10	13	8	12	12
25-29	10	9	9	8	12
30-34	8	9	7	7	8
35-39	5	6	8	6	7
40-44	5	4	4	4	5
45-49	4	6	3	7	5
50-54	4	4	4	5	3
55-59	5	4	3	3	2
60-64	3	5	2	3	2
65-69	1	1	1	2	1
>69	2	0	3	2	2

The proportion (in five yearly age groups) of each community sample is expressed as a percentage of the total number of 5-84 year olds in each community.

The column titled Australian Bureau of Statistics (ABS) shows the corresponding estimate for the Northern Territory Aboriginal population aged 5-84 years in 1986 (55) . The ABS statistics were adjusted so that the denominator was those aged 5-84 years only.

Figure 4.1

Stratification of the sample from CY1 by age and gender.

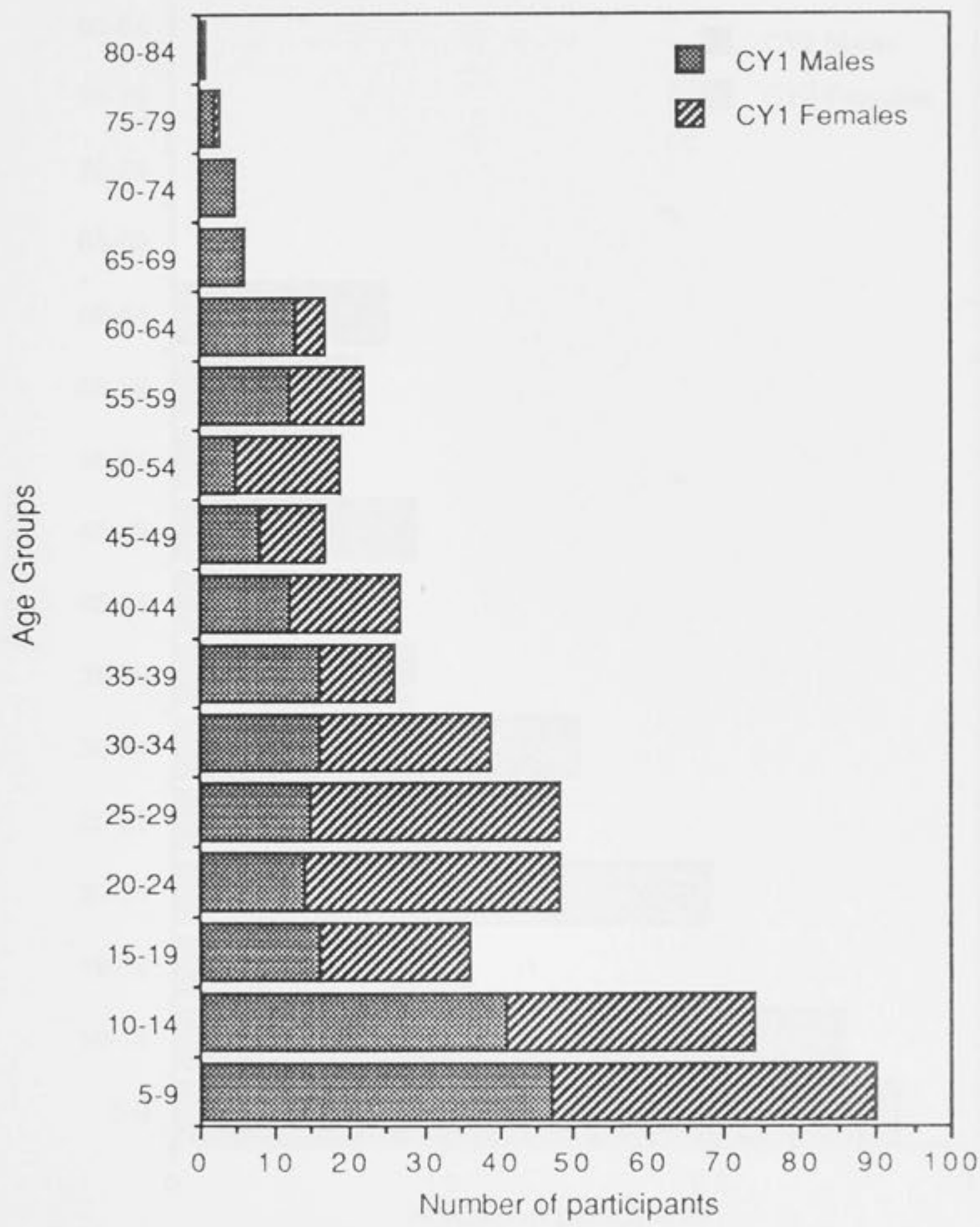


Figure 4.2

Stratification of the sample from CY2 by age and gender.

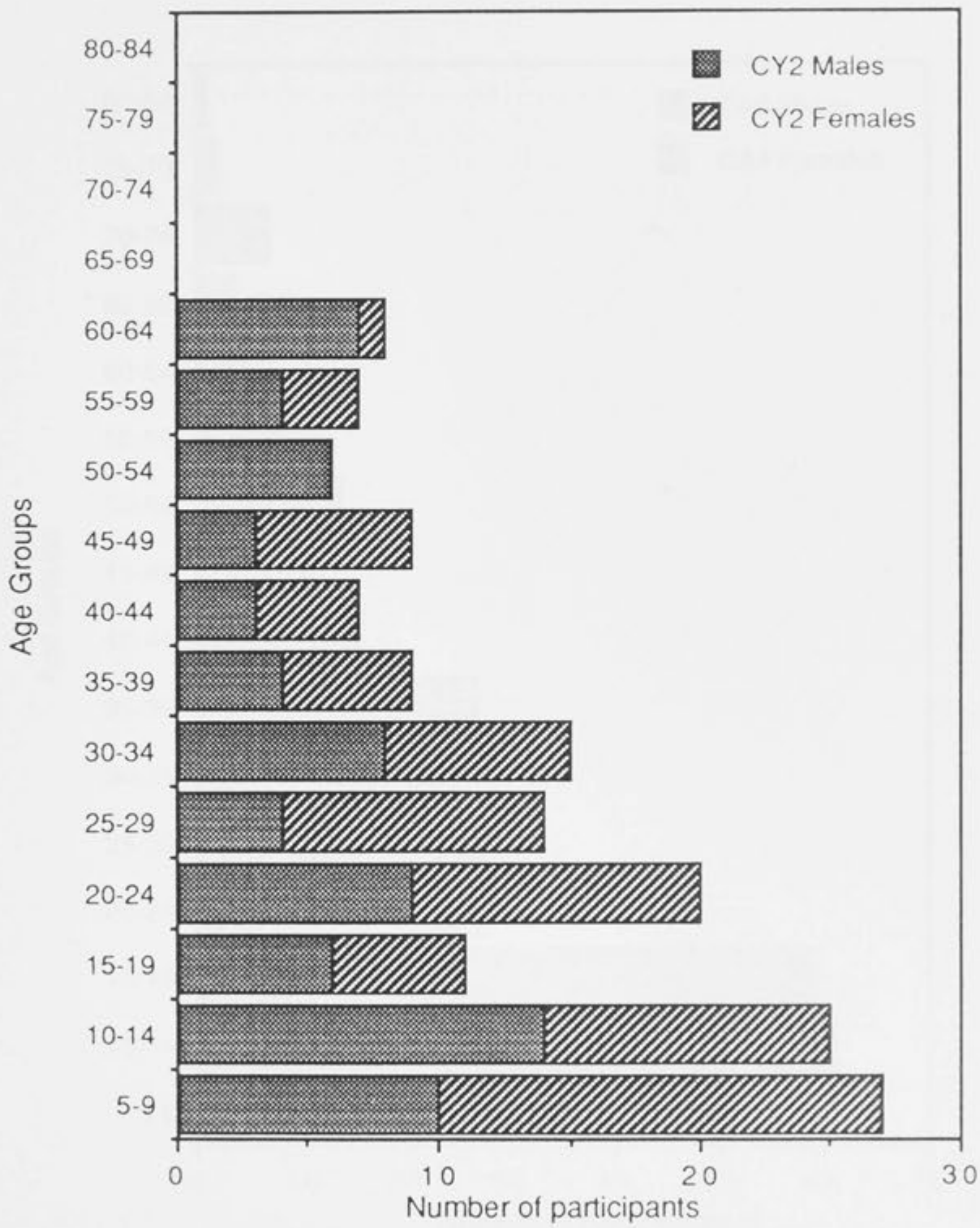


Figure 4.3

Stratification of the sample from CA3 by age and gender.

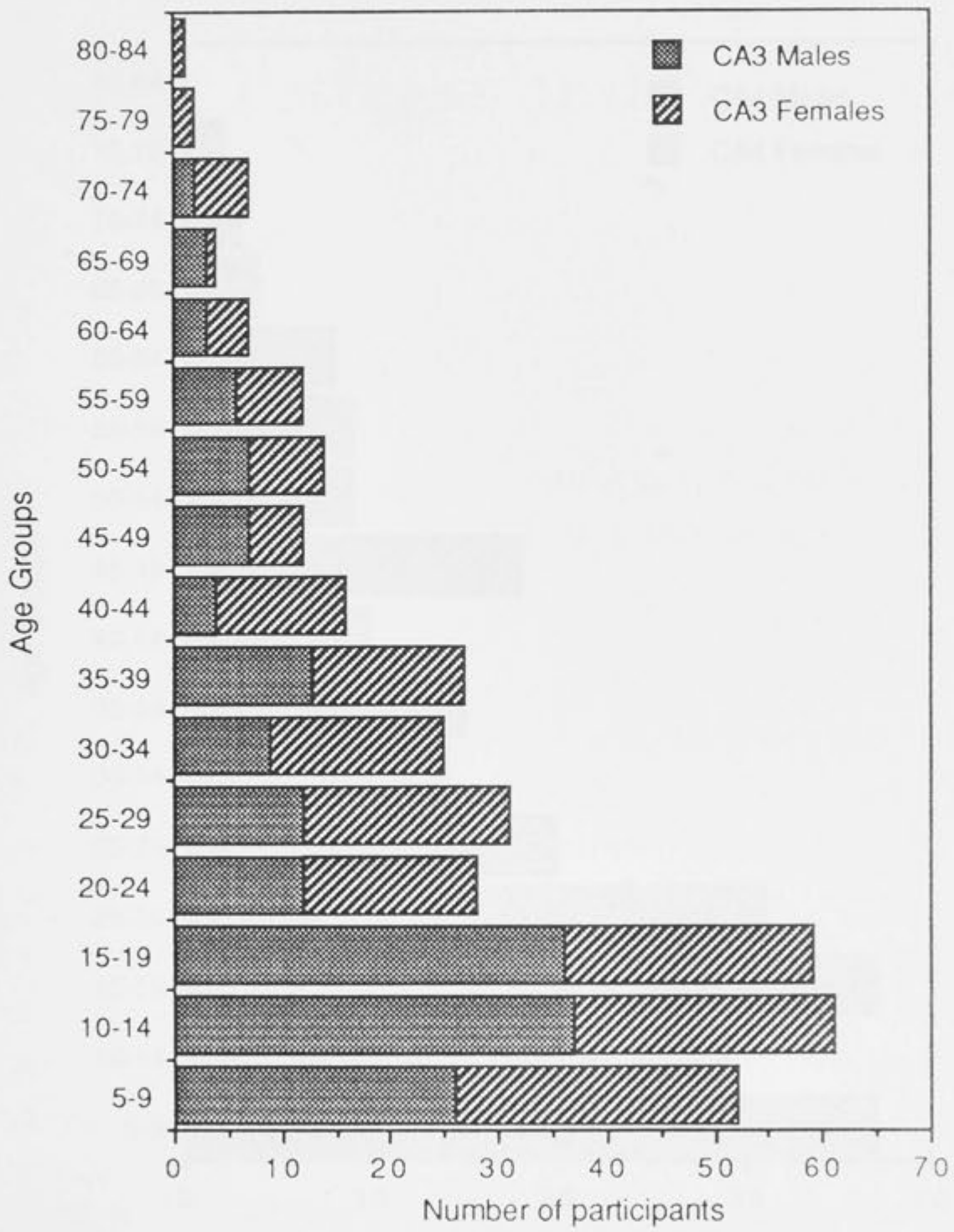
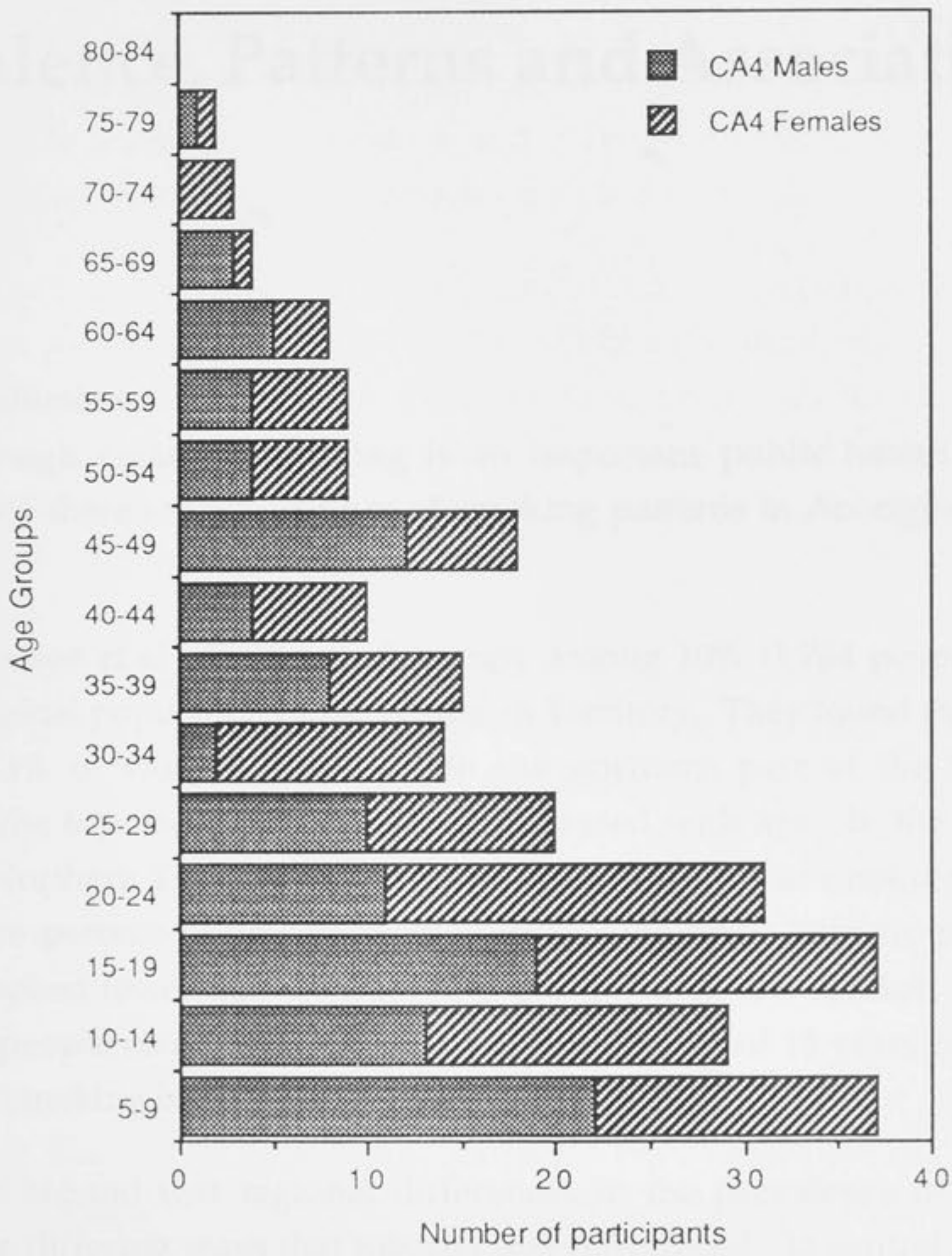


Figure 4.4
Stratification of the sample from CA4 by age and gender.



CHAPTER 5

Cigarette Smoking: Prevalence, Patterns and Associations

5.1 Introduction

Although cigarette smoking is an important public health issue in Australia (56) there are few studies of smoking patterns in Aborigines (15-17, 57).

In 1988, Watson *et al* (17) reported a study among 10% (1,764 people) of the adult Aboriginal population in the Northern Territory. They found that 70% of men and 43% of women smoked. In the northern part of the Northern Territory, "the top end", the prevalence increased with age. In the southern part of the Northern Territory, "the centre", the prevalence of smoking fell with age. Twelve percent of non-smokers were ex-smokers. Fifty-six percent of smokers smoked fewer than 10 cigarettes per day and 40% smoked 11-30 per day. Most people commenced smoking before the age of 15 years but that in the centre, "smoking began later".

Watson *et al* argued that regional differences in the prevalence of smoking reflected the differing ways that tobacco was introduced. In central Australia prior to 1788 tobacco was chewed in the from of "native tobacco" (*Nicotiana*) or Pituri (*Duboisa hopwoodii*). In "the top end" tobacco smoking was probably introduced before 1788 by Indonesian fishermen (58).

Watson *et al* noted that cultural factors as well as addictive factors played an important role in maintaining the high smoking prevalence in the top end (59). In addition most people reported that they liked the taste and the feeling associated with smoking and men reported it relieved boredom. This suggested that pleasure associated with smoking was also an important factor sustaining this behaviour. Non-smokers cited dislike of the taste and health concerns as reasons for not smoking. This suggested that some Aborigines are aware that smoking can be unhealthy and that this knowledge can influence smoking behaviour.

In 1989 Lake (57) reported smoking and alcohol histories from 102 consecutive patients aged 16 years and over attending the Aboriginal medical service in Adelaide. Lake found that 78% of the males and 64% of the females were smokers. There were no pipe smokers and very few "rolled their own". The males smoked an average of 22 cigarettes a day and the females 20.

In 1990 Stephenson (16) reported a study of 210 Aboriginal people (83% of the Aboriginal population aged over 19 years) in Wilcannia Shire, NSW. He found that 71% of the males and 75% of the females smoked. About half the non-smokers were ex-smokers. The prevalence of smoking tended to increase with age in men and to decrease in women. Approximately 10% of smokers smoked fewer than 10 cigarettes daily, 30% smoked 10-19 per day, 35% smoked 20-29 daily, and the remainder smoked more than 30 per day. The ex-smokers were asked their reasons for quitting. Fifty-three percent indicated that they had stopped smoking for health reasons, 16% stopped because they were "sick of smoking", 11% because they "wanted to", 7% because of the expense, 4% because of family pressure and 9% gave other reasons.

In 1992 Guest *et al* (15) reported the prevalence of smoking in 306 adult Aborigines living in a Victorian country town. In males the prevalence was 67% and in females 63%. The prevalence peaked in 25-44 year olds and appeared to decline with age. Twelve percent of males were ex-smokers as were 13% of females. Cigarette consumption was evenly divided in three strata: 1-10 cigarettes per day, 10-20 per day and more than 20 per day.

In 1990 a workshop was held in Perth, WA , on "Preventing Smoking Related Diseases Among Aboriginals" (2, 60). A majority of the participants were Aboriginal or were representatives from Aboriginal medical services. The lack of data regarding smoking in Aborigines was highlighted and evidence was presented (61) which showed there had been no systematic approach to anti-

smoking education in Aboriginal communities. Those at "the workshop" developed recommendations regarding further research.

These recommendations were:

- That surveys be undertaken to assess the prevalence of tobacco smoking and chewing, and that regional and gender differences be documented;
- That morbidity data be collected on smoking related disease, and that attention be directed to assessing the impact of passive smoking on infants and children;
- That studies be undertaken with the object of understanding the aetiology of tobacco smoking and chewing, paying particular attention to socio-cultural and psychological factors. In particular a need for studies that investigate attitudes and beliefs regarding the health effects of tobacco smoking and chewing was identified.

The studies reviewed above suggest that the prevalence of smoking in Aboriginal communities is generally high in comparison with that in non-Aboriginal communities. However there is evidence that the prevalence may be lower in some regions such as central Australia. Regional variation in the volume smoked has also been documented. Urban smokers and smokers in the top end of the NT smoke more than smokers in "the centre". Whilst it has been acknowledged that the cultural aspects* of smoking are likely to be important other factors have been shown to play a role. If the prevalence of smoking is to be reduced in Aboriginal communities then a better understanding of smoking behaviour is required. "The workshop" recommendations highlighted the knowledge gaps in this area and provided a background and rationale for the aims of this component of the present study.

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* Cigarette smoking often occurs in the setting of a gathering of friends or family. Here the sharing of cigarettes (and/or tobacco, alcohol etc) may strengthen social bonds and discharge obligations. An inquiry into these cultural aspects of smoking was beyond the scope of the present study.

5.2 Aims

The aim was:

- To determine the prevalence of smoking by age , region and gender;
- To determine the age at commencement of smoking;
- To determine what people smoke;
- To determine how frequently and how much people smoke;
- To explore the relationship between smoking and tobacco chewing;
- To explore the relationship between alcohol use and smoking;
- To explore the relationship between health knowledge and smoking.

5.3 Methods

Questions to investigate smoking behaviour were developed by the author and incorporated into the study questionnaire. Tidal carbon monoxide was measured to validate smoking status and to focus discussion about the health effects of smoking (see chapter 3).

5.3.1 Questionnaire

The questions (see figure 5.1) were developed following consultation with health workers in CY1 and CY2, Dr Thompson and Dr John Taylor. Dr Taylor is medical anthropologist with experience working with Cape York Aborigines (62). The questions were piloted for comprehensibility and modified where necessary. The same questions were used in all communities.

Q2 was not used in CY1 and CY2 as tobacco was not chewed in these communities. Q6 was an additional question developed for use in CA3 and CA4 to assist those with limited numeracy to estimate their age at smoking commencement. The answers to Q7 were converted to a number of cigarettes smoked daily.

The questions were administered during an interview with the assistance of an Aboriginal translator. All the translators understood the purpose of the questions. Several questions were open-ended to encourage free responses.

Figure 5.1

Questionnaire to assess smoking, tobacco chewing, alcohol use and "knowledge" of the health consequences of smoking.

Q1	Do you smoke?	Yes/No	
Q2	Do you chew tobacco?	Yes/No	
Q3	What do you smoke?	(	)
Q4	Do you smoke every day?	Yes/No	
Q5	How old were you (in years) when you started smoking?	(	)
Q6	How old were you when you started smoking?	Kid	0-13
		Young Fella	14-19
		Married	>20
		Don't Know	
Q7	How many cigarettes (or packs) do you smoke a day?	(	)
Q8	How many tins of tobacco do you smoke each week?	(	)
Q9	Do you know of any sickness caused by smoking?	Yes/No	
Q10	If yes (to Q9) then what?	(	)
Q11	Do you drink alcohol?	Not at all	
		A little bit	
		A moderate amount	
		Heavily at times	
		Every day	

*For non-smokers:

Q12	Why don't you smoke?	(	)
Q13	Did you ever smoke regularly?	Yes/No	
Q14	If yes (to Q13) why did you give up?	(	)

5.3.2 Tidal Carbon Monoxide

Tidal carbon monoxide was measured (blind to the participant's smoking status) using a recently calibrated Bedfont EC50 Smokerlyser (63). After the reading was completed the author asked each participant about their smoking status. During the ensuing discussion information about the health effects of smoking was shared with all participants and smokers were encouraged to quit. The tidal carbon monoxide level was expressed in parts per million (ppm) and the results were expressed as the mean and standard deviation for each of several subgroups (by age and gender) of the population.

5.3.3 Analysis

The responses to the open ended questions (Q10, Q12 and Q14) were arranged into nominal groups by the author after an assessment of the range and nature of responses. All frequency data were tabulated with the number (N) of eligible subjects and the number of missing values (MV). Binomial dummy variables were created to distinguish those that drank no alcohol from those that drank any amount of alcohol. Similarly, dummy variables were created to distinguish those that had no health "knowledge" regarding the consequences of smoking from those that had any. Two by two cross-tabulations were performed to examine the associations between several variables and unadjusted odds ratios were calculated to quantify the relationships.

5.4 Results

5.4.1 Prevalence and Commencement of Smoking

The prevalence of smoking among 15-84 year old participants ranged from 51 to 75% in men and 1 to 78% in women (see table 5.1). In all communities smoking was rarely reported under the age of 14 years. The prevalence of smoking peaked in 25-44 year old males and 15-24 year old females. In CA4 (males and females) and CY1 females the prevalence of smoking fell significantly with age (chi-square trend $p<0.01$). Among males there was no significant difference in the prevalence of smoking between the communities but among females there were significant differences (chi-square $p<0.001$). The prevalence of smoking in CA3 and CA4 women was relatively low, indeed in CA3 there was only one female smoker. When these data from all communities were combined the prevalence of smoking was higher in men than women. In addition the prevalence of smoking among men was relatively

stable with age but fell significantly with age in women (chi-square trend $p<0.001$).

Q5 showed that most smokers commenced smoking between the ages of 14 and 18 years (see table 5.2). In CA3 and CA4 the prevalence of missing values was relatively high for this question. There were a similar number of missing values for Q6 in CA4 but fewer in CA3. In CA3 and CA4 the findings for Q6 concurred with those of Q5 and suggested that the majority of people commenced smoking during the mid- to late teenage years (see table 5.3).

5.4.2 Smoking Pattern and Volume

All smokers in CY1, CY2 and CA3, and 98.5% in CA4 smoked cigarettes. Less than 10 percent of smokers rolled their own and less than 2% smoked pipes.

The proportion of smokers smoking daily ranged from 42% in CA3 to 86% in CY2 (see table 5.4). There were no significant gender differences but daily cigarette consumption was significantly more prevalent among the Cape York smokers ($p < 0.001$).

Table 5.5 shows the approximate number of cigarettes smoked daily by users. In Cape York 15% of smokers smoked 1-5 cigarettes per day and 45% smoked 16-30 per day. In central Australia 69% smoked 1-5 cigarettes per day. Less than 12% of smokers in any community smoked more than 30 per day. There were no significant gender differences in the quantity of cigarettes smoked.

5.4.3 Tidal Carbon Monoxide in Smokers and Non-smokers

The mean tidal carbon monoxide level in non-smokers was 4 ppm (range 1-23, SD 2 ppm). The mean tidal carbon monoxide level in smokers was 14 ppm (range 2-43, SD 8 ppm). Twenty-seven people who described themselves as non-smokers had tidal carbon monoxide levels which were discrepant (more than 2 standard deviations above the non-smoking mean). The highest mean levels of tidal carbon monoxide (16 ppm) occurred in the 25-44 year old smokers. The mean number of cigarettes smoked each day was 18 (SD 15) with a range of 1 to 75. The correlation between the number of cigarettes smoked and the tidal carbon monoxide was significant ($p < 0.0001$) but weak (Pearson Correlation Coefficient 0.258).

5.4.4 Tobacco Chewing and Cigarette Use

Table 5.6 shows that in CA3 and CA4 34% of men and approximately 60% of women chewed tobacco. Table 5.7 shows the relationship between

tobacco chewing and tobacco smoking. The odds ratio for the association was 0.34 (95% CI 0.22-0.54). The relationship between tobacco chewing and smoking did not differ by gender.

5.4.5 Alcohol Consumption and Cigarette Use

Table 5.8 details self-reported alcohol consumption in subjects over the age of 14 years. In males the prevalence of non-drinkers (teetotallers) ranged from 13% to 49%. In women the prevalence of teetotallers ranged from 40% to 91%. In both sexes the heaviest levels of self-reported alcohol consumption came from CY2 and the lowest levels were from CA3.

Table 5.9 shows a cross-tabulation between alcohol consumption and cigarette smoking status. All subjects were coded as smokers or non-smokers and drinkers or non-drinkers using the answers to Q1 and Q11. Forty percent were non-smoking teetotallers, 14% were smoking teetotallers, 14% drank alcohol but did not smoke and 32% both drank and smoked. The odds ratio for the association between smoking status and alcohol consumption was 5.74 (95% CI 4.25-7.69). Stratification by community and gender did not reveal any regional or gender differences in the relationship between alcohol and cigarette consumption.

5.4.6 Smoking and Health Knowledge

Table 5.10 shows the regional variation in the prevalence of participants with "knowledge" of the health consequences of smoking. Subjects were coded as "knowledgable" if they answered yes to the question (Q9) "Do you know of any sickness caused by smoking?". In CY1 73% of subjects over the age of 14 were aware of at least one adverse health effect of smoking. In CA3 and CA4 less than 30% were "knowledgable". The prevalence of missing values for this question was 23% in CY1 and 56% in CY2. Overall the prevalence of missing values was 20% in non-smokers and 16% in smokers. However in CY1 the prevalence of missing values was 44% in non-smokers and 8% in smokers.

Table 5.11 provides details of the illnesses that "knowledgable" participants associated with smoking. The numbers of "knowledgable" participants in CY2 and CA4 were low (<40). Forty-seven to 88% of "knowledgable" participants were aware that smoking was associated with lung cancer. Two to 9% were aware that smoking was associated with heart disease. Four to 26% associated illnesses not conventionally attributed to smoking. In CA3 and CA4 7% and 31% respectively recognised that lung diseases other than cancer could result from smoking.

When these data from the four communities were combined and a cross-tabulation between smoking status and knowledge status was performed (both variables dichotomised 0/1) there was no relationship between individual knowledge and current smoking status. However table 5.12 shows that in CY1 there was a modest reduction in the odds for being a current smoker among "knowledgable" participants (odds ratio 0.35, 95% CI 0.18-0.79).

Table 5.13 lists the reasons non-smokers gave for not smoking (Q12). In all communities the most prevalent reasons given for not smoking were: "I don't like it" or "because it makes you sick". Very few non-smokers attributed their non-smoking status to either the cost of cigarettes or medical advice. The prevalence of missing values was very high in CY2 and CA3. In CY2 the missing values were evenly distributed between men and women. In CA3 115 of the 131 missing values were from women. The high prevalence of missing values in these women reflected a problem with the question. In the field it soon became apparent that the question was not appropriate for the CA3 women because there were strong cultural restraints on women smoking in that community. "Just don't smoke" or blank puzzled looks were common responses to initial attempts to administer this question. With the encouragement of the Aboriginal assistants, the author withdrew this question for women in that community.

The prevalence of ex-smokers among non-smokers is shown in table 5.14. It shows that 21 to 51% of non-smokers were ex-smokers (except CA3 women). Table 5.15 lists the reasons given by ex-smokers when asked why they gave up smoking (Q14). There were 94 ex-smokers of whom 82 answered the question. In CY1, "got chest problems" and "don't like it" were the reasons given by approximately 40% of ex-smokers. A further 15% of ex-smokers in CY1 reported that they stopped smoking because of medical advice and 11% stopped because of the cost of cigarettes. The numbers of ex-smokers in CY2, CA3 and CA4 were small. Ex-smokers in these communities appeared to give up because they "didn't like smoking" or because "it makes you sick".

Table 5.1

Prevalence of smoking by age, gender and community.

Male	CY1		CY2		CA3		CA4		All	
Years										
10-14	0%	0/41	0%	0/13	2.7%	1/37	0%	0/13	1%	1/104
15-24	79%	22/28	57%	8/14	50%	24/48	68%	21/31	62%	75/121
25-34	58%	18/31	83%	10/12	86%	18/21	67%	8/12	71%	54/76
35-44	75%	21/28	86%	6/7	53%	9/17	58%	7/12	67%	43/64
45-54	61%	8/13	67%	6/9	50%	7/14	33%	5/15	51%	26/51
>54	67%	26/39	91%	10/11	43%	6/14	8%#	1/12	57%	43/76
15-84	68%	95/139	75%	40/53	56%	64/114	51%	42/82	63%¤	241/388
Female										
Years										
10-14	3%	1/33	10%	1/10	0%	0/24	6%	1/17	4%	3/84
15-24	74%	39/53	75%	12/16	0%	0/38	42%	17/38	47%	68/145
25-34	68%	38/56	71%	12/17	0%	0/35	18%	4/22	42%	54/130
35-44	24%	6/25	89%	8/9	0%	0/26	29%	4/14	24%	18/74
45-54	30%	7/23	83%	5/6	8.3%	1/12	0%	0/12	25%	13/53
>54	40%#	6/15	86%	6/7	0%	0/19	0%†	0/15	21%#	12/56
15-84	56%	96/172	78%	43/55	1%	1/130	25%	25/101	36%*	165/458

Missing Values = 9

¤ Chi-square for the difference between communities not significant.

* Chi-square for the difference between communities $p < 0.001$

† Chi-square trend for the change in prevalence with age $p < 0.01$

Chi-square trend for the change in prevalence with age $p < 0.001$

Table 5.2

Age of commencement of smoking (Q5) as the cumulative percentage.

	CY1	CY2	CA3	CA4
N	191	83	65	67
MV	1	18	25	32
Age in years	%	%	%	%
12	3	8	7	7
14	11	9	7	31
16	47	26	34	57
18	78	53	68	81
20	88	70	90	98
22	94	88	92	100
30	98	97	98	100

N = number of smokers over the age of 14 years

MV = number of smokers that failed to answer this question (missing values)

Table 5.3

Age of commencement of smoking (Q6) expressed as the cumulative percentage.

	CY1	CY2	CA3	CA4
N	191	83	65	67
MV	-	-	11	32
Age in Years				
0-13	-	-	5	4
14-19	-	-	84	82
>20	-	-	100	100

N = Number of smokers over the age of 14 years

MV = number of smokers that failed to answer this question (missing values)

Table 5.4

Proportion of smokers smoking daily by community in 15-84 year olds.

	CY1	CY2	CA3	CA4
N	191	83	65	67
MV	4	10	3	2
% Smoking daily *	85	86	42	54

N = Number of smokers over the age of 14 years.

MV = number of smokers that failed to answer this question (missing values).

* Chi-square for the significance of the difference between communities $p < 0.001$

Table 5.5

Number of cigarettes smoked each day (by community) expressed as the percentage of smokers in each stratum of usage.

	CY1	CY2	CA3	CA4
N	191	83	65	67
MV	5	37	25	6
Cigarettes /day	%	%	%	%
1-5	13	20	70	68
6-15	30	26	17	11
16-30	50	43	13	17
>30	7	11	0	4

N = Number of smokers over the age of 14 years.

MV = number of smokers that failed to answer this question (missing values).

Table 5.6

Prevalence of tobacco chewing in 15-84 year olds by community.

	CY1*	CY2*	CA3	CA4
Male N	141	56	114	85
MV			4	0
% chew tobacco	0	0	34	34
Female N	173	53	131	102
MV¥			0	0
% chew tobacco	0	0	56	71

N = number of participants.

MV = number that failed to answer this question (missing value) .

* Q2 not required as tobacco chewing not practised in these communities.

Table 5.7

Relationship between tobacco chewing and cigarette smoking in 15–84 year olds in CA3 and CA4.

	Tobacco Smoker	
Tobacco Chewer	No	Yes
No	128	88
Yes	169	40

N=432

Missing Values = 7

Odds Ratio for the association between tobacco chewing and cigarette use 0.34 (95% CI 0.22–0.54).

Table 5.8

Self-reported alcohol consumption in 15-84 year olds by community as a percentage in each community.

	CY1	CY2	CA3	CA4
Male (N)	141	56	114	85
MV	4	8	1	1
Not at all*	33	13	49	43
A little bit	27	25	42	26
Moderately	28	37	1	11
Heavy at times	9	23	6	19
Every day	3	2	2	1
Female (N)	173	53	131	102
MV	1	5	1	0
Not at all*	60	40	91	61
A little bit	31	33	8	22
Moderately	8	17	0	7
Heavy at times	1	10	1	10
Every day	0	0	0	0

* Chi-square for the significance of the difference between communities $p < 0.001$

Table 5.9

Relationship between alcohol consumption and cigarette use in 15-84 year olds.

	Smoking	
	No	Yes
Alcohol Drinker		
No	319	124
Yes	124	277

N=855

Missing Values = 11

Odds Ratio for the association between alcohol and cigarette use 5.74 (95% CI 4.25-7.69)

Table 5.10

Prevalence of "knowledgable" participants by community.

	CY1	CY2	CA3	CA4
N	314	109	245	187
MV	71	61	18	13
N "Knowledgable"	177/243	21/48	63/227	39/174
% "Knowledgable"*	73	44	28	22

N = Number of participants over the age of 14 years.

MV = Number of participants that failed to answer this question (missing values).

* Chi-square for the significance of the difference between communities $p < 0.001$

Table 5.11

Details of the illnesses that "knowledgable" participants" associated with smoking. Results expressed by community as a percentage of those that answered the question.

	CY1	CY2	CA3	CA4
N	177	21	63	39
MV	1	0	9	0
Lung Cancer	88	57	57	47
Heart Disease	4	9	6	2
Both the above	4	29	4	5
Other**	4	5	26	15
Lung Disease***	0	0	7	31

N = number of "knowledgable" participants"

MV = number of "knowledgable" participants that failed to answer question (missing values)

** colds tuberculosis headaches "get sick"

***Lung Disease ± Lung Cancer

Table 5.12

Relationship between "knowledge" of the health consequences of smoking and current smoking status in CY1 15-84 year olds.

	Current Smoker	
	No	Yes
"Knowledgable"		
No	10	56
Yes	58	119

N=314

Missing Values = 71. The prevalence of missing values in non-smokers was 44% and in smokers 8%.

Odds Ratio for the association between "knowledge" of the health effects of smoking and current smoking status 0.36 (95% CI 0.18-0.79)

Table 5.13

Reasons for not smoking given by 15-84 year old non-smokers (by community) expressed as a percentage of those that answered the question.

	CY1	CY2	CA3	CA4
N	120	25	179	116
MV	39	20	154**	25
"Don't like it"	63	80	72	43
"Makes you sick"	12	20	24	44
Other †	25	0	4	11

N = Number of non-smokers in each community

MV = Number of missing values

** 115 of missing values are female non-smokers

† Includes, high cost of cigarettes, advice not to smoke, "caused coughing" and "unsure"

Table 5.14

Prevalence of ex-smokers in 15-84 year old non-smokers, by community expressed as a percentage.

	CY1	CY2	CA3	CA4
N	120	25	179	116
MV	21	9	131**	44
% Ex-smokers*	51	50	42	21

N = number of non-smokers in each community.

MV = number of missing values.

** 115 of missing values are female non-smokers.

* Chi-square for the significance of the difference between communities $p < 0.001$

Table 5.15

Reasons for giving up smoking (Q14) in 15-84 year old ex-smokers, by community expressed as a percentage .

	CY1	CY2	CA3	CA4
N	51	8	20	15
MV	4	3	4	1
"Got chest problems"	19			
"Don't like it"	19	40	50	
"Makes you sick"			43	64
Advised not to smoke	13			15
High cost of cigarettes	11	40		
Other "unsure"†	38	20	7	21

N = number of ex-smokers in each community.

N = number of missing values.

† Includes subjects who gave up because of coughing which they attributed to smoking

5.5 Discussion

The prevalence of smoking in the two Cape York communities was similar to that found among white Australian males after World War Two (56) (see table 5.16). This finding concurred with the reports by Watson *et al* , Lake and Stephenson (see table 5.16). The lower prevalence in central Australia was also consistent with Watson *et al's* observations and the remarkably low prevalence in CA3 women has been reported previously by Gault (32). The fall in the prevalence of smoking with age among men from CA4 and among women in CY1 and CA4 suggested that the prevalence of smoking was rising in these communities as teenagers commenced smoking (a cohort effect). There appears to be an opportunity to prevent smoking becoming entrenched in central Australia (especially among women) if successful measures to reduce the up-take of smoking in teenagers can be instituted.

Table 5.16

Prevalence of smoking in Aboriginal and non-Aboriginal Australian communities presented as a percentage of the populations studied.

	Year	Male	Female	Reference
Non-Aboriginal	1945	72	26	(56)
Non-Aboriginal	1983	37	30	(56)
Top End* NT	1986	81	72	(17)
Katherine* NT	1986	71	35	(17)
Centre* NT	1986	59	9	(17)
Adelaide* SA	1989	78	64	(57)
Wilcannia * NSW	1990	71	75	(16)
CY1*	1990	68	56	
CY2*	1990	75	78	
CA3*	1991	56	1	
CA4*	1991	51	25	

* Aboriginal Communities

These data presented in tables 5.1, 5.2 and 5.3 show that smoking initiation occurs around 14 years of age. This is younger than reported by Watson *et al* and may indicate a trend towards earlier commencement of smoking. Q6, the age for commencement of smoking question designed for CA3 and CA4 increased the response rate in CA3 but had no effect in CA4. The high number of missing values highlighted the difficulty of asking quantitative questions in communities where most people have limited numeracy. Overall the findings indicated that interventions to reduce the initiation of smoking will need to target 10-13 year old children.

Smoking patterns differed significantly between Cape York and central Australia (see tables 5.4 and 5.5). In Cape York most smokers were smoking more than six cigarettes every day. In central Australia approximately 50% of smokers did not smoke every day and less than 30% of smokers smoked more than 5 cigarettes per day. These observations were important because people smoking infrequently are less likely to be addicted to nicotine than regular smokers. It is unlikely that cigarette smokers in CA3 and CA4 were obtaining

nicotine from tobacco chewing when cigarettes were not available because these different modes of tobacco use were essentially mutually exclusive (see table 5.7). These findings suggest that smoking intervention programs in CY1 and CY2 will need to incorporate strategies for managing nicotine withdrawal but in the central Australian communities nicotine withdrawal is less likely to be a problem.

Most smokers in CA3 and CA4 smoked intermittently and even then they only smoked 1-5 cigarettes a day. Possible explanations for both the irregularity of smoking and the relatively small numbers of cigarettes consumed are that community members only smoked when they felt like it or they were opportunistic smokers, smoking only when cigarettes were available. Personal access to cigarettes was not assessed in this study although the author noted that cigarettes were readily available from stores in all four communities*.

The tidal carbon monoxide recordings suggested that most people answered the smoking question accurately. The correlation (Pearson R value 0.26) between the number of cigarettes smoked daily and tidal carbon monoxide levels suggested that these data regarding the number of cigarettes smoked had some validity. In men the prevalence of smoking and the mean tidal carbon monoxide levels both peaked in 25-44 year olds. This suggested that the frequency of smoking was highest in younger smokers.

The prevalence of tobacco chewing in CA3 and CA4 and the relationship to smoking were similar to the findings of Watson *et al* . In the centre, tobacco chewing was the most prevalent form of tobacco consumption. It is not known to what extent people may shift from being principally tobacco chewers to tobacco smokers and vice versa. Although tobacco chewing is known to be associated with oropharyngeal cancers, the relative dangers of tobacco smoking versus chewing have not been investigated in Aborigines. Tobacco chewing in these communities could be suggested as a culturally acceptable alternative for those heavier smokers wanting assistance to quit.

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* In CA4 at the time of the data collection the new non-Aboriginal store manager had just completed the installation of a cigarette vending machine in the foyer of the store. This gave children with money ready access to cigarettes. In the author's experience store managers in Aboriginal communities often make decisions based on economic imperatives with little regard for the public health consequences of the decisions.

These data regarding alcohol consumption were collected to examine associations with smoking (see table 5.9). The scale for alcohol consumption combined notions of frequency and volume (see table 5.8). It was interesting to note that the regional variation in self-reported alcohol consumption reflected access to alcohol. Access to alcohol by community in descending order of availability was CY2, CY1, CA4 and CA3. CY2 with ready alcohol availability had the highest self-reported levels of consumption and CA3, with the most restricted access had the lowest levels. This observation was consistent with Watson *et al's* conclusion that, "In all locations, community types and liquor status's (sic), it appears that the more available alcohol was, the higher the percentage of drinkers and the more often they drank". The present study highlights the importance of supporting Aboriginal efforts to control access to alcohol in their communities.

The relationship between alcohol consumption and cigarette smoking was strong (see table 5.9). Watson *et al* found a similar association. Cigarette smoking and alcohol drinking are activities that often occur when friends and relatives meet. Cognisance of the relationship between the use of these two substances and an understanding of their wider social role is necessary if programs designed to reduce their use are to be successful.

The question seeking knowledge of the health consequences of smoking (Q9) suffered from a design problem that meant that if the instructions were followed only smokers were asked this question. This fault was recognised prior to the field work so the questionnaire teams were asked to ignore the errant instructions. However the responses from CY1 suggested that the revised instructions were not always followed (see section 5.4.6). In CY2 we had different Aboriginal assistants each day and many, not having a health background, had difficulty understanding the purpose of Q9. This contributed to the low response rate for the question (see table 5.10). As a result of the two problems described above the present study had fewer responses for Q9 from non-smokers in CY1 and both smokers and non-smokers in CY2. This reduced the power of the study to examine the regional variation in the prevalence of knowledge and the relationship between "knowledge" and smoking status.

Despite the problems described above, significant variation in the prevalence of "knowledgable" participants was detected (see table 5.10). Interpreting these differences was complicated by the cultural, language and educational diversity between the communities. Nevertheless these data suggest that a

high proportion of the population in CY1 had some awareness of the dangers of smoking and that very few in CA3 and CA4 displayed this knowledge.

The reasons for the variation in prevalence of "knowledgeable" participants are not known but it was probably significant that "knowledge" was most prevalent in CY1 where English literacy was relatively common. It is likely that English literacy allows Aboriginal people to be influenced by the health messages written on cigarette packets and other health promotion material designed for the wider Australian population. Hill *et al* (56) in an Australia wide survey of 5,580 adults (age 16 and over), found that only 14% of the sample were unaware that smoking was a health hazard. Hill *et al* also found that the higher an individuals educational level the more likely illness was attributed to smoking. In adults with only primary school education nearly a third did not believe smoking was unhealthy. The present study suggests that in Aboriginal communities there is a relationship between educational attainment and health knowledge.

The relative frequency of specific conditions attributed to smoking (see table 5.11) was similar to that found by Hill *et al*. In Hill *et al's* study it can be deduced that 81% of "knowledgable people" were aware smoking could cause lung cancer, 24% other cancers, 27% heart disease, 27% bronchitis, 24% emphysema and 9% associated smoking with strokes and other vascular disease. It is interesting that lung cancer was the most frequently recognised disease to be associated with smoking in both Hill *et al's* and the present study. This observation suggests that health messages regarding the hazards of smoking have had some impact in the study communities. However the findings suggest that the educational messages recalled by Aboriginal people have focussed on lung cancer to the exclusion of the other associations. This emphasis should be changed because diseases other than lung cancer (such as ischaemic heart disease) are numerically more frequent consequences of smoking and thus have greater public health importance (64) .

In the present study there was no overall relationship between "knowledge" and smoking status. However when the data for CY1 were examined independently there was a significant inverse relationship (see table 5.12). This suggested that even though knowledge was very limited there may have been a relationship between health knowledge and smoking behaviour in CY1. Although the effect was not strong, the observation suggests that health promotion initiatives designed to increase Aboriginal awareness of the dangers of smoking have the potential to reduce the prevalence of its use.

The reasons given by never-smokers and ex-smokers for not smoking were similar and frequently interrelated as many people stated that they "didn't like it" because "it makes you sick" (see figures 5.14 and 5.15). The observation that 20-50% of non-smokers were ex-smokers (except among women in CA3 where none were) suggests that each community had a small but significant core of ex-smokers. A strong cultural barrier to smoking initiation was evident among CA3 women. The present study indicated that Aboriginal people chose not to smoke or quit smoking because either it was not culturally acceptable, they didn't like it, or because it was perceived to be unhealthy.

It has been shown by Hill *et al* in the general Australian population that, "a predisposition to quit smoking can be induced by increasing smoker's beliefs that smoking causes disease, and by raising the perceived probability that their smoking will lead to a fatal illness" (56). Hill *et al* also noted that, "Our findings indicate it is not true that there is not a child or adult in Australia who does not know that cigarette smoking is bad, and that neither is it true that all smokers take a risk they fully understand, nor that basic health education in these matters is redundant". This present study suggests that Hill *et al's* conclusions are also relevant for Aboriginal people.

Discussions revealed that it was not self evident to people living in these small communities that smoking is dangerous. This may be because the absolute risk of dying from lung cancer (the most commonly identified adverse effect of smoking) in smokers is approximately 140/100,000/year (64). In an Aboriginal community of 330 smokers, this rate would manifest as one death every two or three years. However, the absolute risk of dying from ischaemic heart disease in smokers is 669/100,000/year (64). This would manifest as 2 deaths per year in a community of 330 smokers. The high prevalence of chronic lung disease in Aboriginal communities (see chapter 9), together with the high prevalence of hypertension, diabetes and obesity which are also risk factors for heart disease (28), means that the mortality attributable to smoking in Aboriginal communities undoubtedly exceeds 669/100,000 per year. If Aboriginal people linked smoking with death from ischaemic heart disease, the adverse consequences of smoking would be more frequently observed as deaths from this cause are relatively common in Aboriginal communities.

5.6 Conclusion

- The prevalence of smoking among males and females in Cape York and among males in central Australia was high when compared with that of non-Aboriginal Australians. In comparison with non-Aboriginal Australians the prevalence of smoking among women in central Australia was low.

- The prevalence of smoking among some groups appeared to be rising due to high smoking initiation rates among teenagers.

- Most smokers in Cape York consumed more than 15 cigarettes daily whereas in central Australia most smoked fewer than 6 cigarettes daily.

- Younger smokers were probably smoking more frequently than older smokers.

- In central Australia tobacco chewing and smoking were usually mutually exclusive habits.

- There was a strong association between cigarette smoking and alcohol consumption.

- Expressed knowledge of the health consequences of smoking was limited.

- In CY1 knowledge of the health consequences of smoking was associated with a reduction in the odds for being a current smoker.

- Not liking smoking or concern about the health consequences were common explanations for never smoking or for quitting smoking.

- There is an urgent need for appropriately designed educational programs in Aboriginal communities to significantly reduce the prevalence and uptake of smoking.

CHAPTER 6

Asthma and Atopy

6.1 Introduction

The literature suggests that the prevalence of asthma in Aborigines is less than 4% (see chapter 2). This is low when contrasted with the prevalence of asthma in non-Aboriginal Australians (38, 46).

There is evidence to suggest that exposure to house dust mite allergen (*Der p*1) may be important in the genesis of asthma (29, 65), and levels of *Der p*1 above 10 micro grams/gram of fine dust have been shown to cause sensitisation and symptoms (66). House dust mites vary in prevalence throughout Australia because they require humidity above 55% to flourish (67). The present study provided an opportunity to study the role of house dust mite allergy in the aetiology of asthma in Aborigines.

The role of genetics in the development of atopy and asthma remains uncertain. Dr Thompson observed that CY1, a community with a history of much greater contact with European and Chinese people than CY2, had a relatively high prevalence of asthma compared with CY2. He had not identified any Aboriginal asthmatics in CY2. Dr Thompson hypothesised that asthma and or atopy in CY1 could be linked to introduced genes.

Allergic or atopic people produce immunoglobulin E (IgE) in response to ingested or inhaled allergens. The prevalence of atopy is known to increase with age until approximately the age of 24, and serum IgE levels parallel this rise (68). Atopy has been shown to be associated with asthma (69). The

relationships between asthma, atopy and elevated IgE have not been investigated in Aborigines.

Bronchial hyperresponsiveness (BHR) is an airway abnormality associated with asthma and chronic obstructive pulmonary disease (70). BHR may be reliably detected by measuring the changes in FEV_1 that occur following the inhalation of histamine (30). BHR in conjunction with recent wheezing is an accepted definition for asthma in epidemiological studies (71).

The need for research relating to asthma is recognised in the National Aboriginal Health strategy (28).

6.2 Aims

To determine the prevalence of asthma and its associations in adults and children by determining the prevalence of:

- Ever wheeze and recent wheeze;
- Bronchial hyperresponsiveness;
- Atopy to common aero-allergens;
- House dust mite allergen exposure;
- Elevated total IgE in asthmatics and controls (CY1 and CY2);
- Non-Aboriginal HLA DR and DQ alleles in asthmatics and controls from (CY1 and CY2).

6.3 Definitions

The following definitions have proven utility in studies of the epidemiology of asthma (71):

•Recent wheeze (RW)	A history of wheeze in the last 12 months.
•Ever wheeze	A history of wheeze at any time since birth.
•$PD_{20}FEV_1$	Dose of histamine required to produce a 20% fall in FEV_1 during a histamine challenge test.
•BHR	$PD_{20}FEV_1$ less than or equal to 3.9 µmols of histamine.

•Current asthma	The coexistence of recent wheeze and BHR; or recent wheeze and a greater than 15% increase in FEV_1 following bronchodilator.
•Atopy	A wheal reaction of 3mm or more to a skin test using standardised allergens.

6.4 Methods

All subjects aged five years and over answered a respiratory symptom questionnaire that included questions about ever and recent wheezing. In addition, all subjects were examined for rhonchi (blind to the questionnaire responses). Skin tests to common allergens and a histamine challenge test were also performed. In CY1 and CY2 blood was collected from 96 people (all asthmatics and selected controls) for estimation of total IgE and HLA DR sub-typing.

Inter-observer agreement regarding the presence of rhonchi was assessed to validate the rhonchi observations. The association between rhonchi and recent wheezing was examined because one would predict that rhonchi would be more prevalent in subjects with a history of recent wheeze than in those without. A significant association between "recent wheeze" and rhonchi would imply the "recent wheeze" question had predictive validity. The association was assessed by calculating an odds ratio and the sensitivity and specificity of rhonchi for recent wheeze.

6.4.1 Questionnaire

The questions regarding wheeze are listed in Table 6.1. The questions were developed in consultation with health workers from CY1 and CY2, and they were piloted in all four communities. In all communities (except CY2 where there were no known Aboriginal people with asthma), health workers were familiar with the terms wheeze and asthma. In all communities the questionnaire was administered by a pair comprising a local Aboriginal health worker (or community member) and a member of the technical staff. Members of the technical staff demonstrated wheeze to assist interpretation of the questions. In CA3 and CA4 a tape recording of wheezy breathing was also used to assist the history taking. The tape was not available when data were collected from CY1 and CY2. Generally children answered the questions with an adult family member or guardian present.

Table 6.1
Asthma Questions

Question
Some people have wheezy, noisy breathing from time to time. Are you like this?
If yes to wheeze question then..Was it in the last 12 months?

6.4.2 Skin Tests

To assess the prevalence of atopy, skin prick tests to common allergens were performed using the method of Pepys (72). The arm was initially cleaned and a white grid was applied to the forearm to facilitate identification of the allergens. A small drop of each allergen was placed in the grid and a sterile lance was passed through the droplet into the epidermal skin layer. The lance was wiped clean of allergen with an alcohol swab between each prick. The arm was then blotted dry and the time of testing was recorded on the arm. The skin tests were read 15 minutes after application of the allergens. The length of the longest axis of the wheal and the perpendicular were recorded. Histamine 1mg/ml was used as a positive control and saline was used as a negative control.

In Cape York the following allergens were used: cockroach, house dust, *Dermatophagoides farinae* (DF), *Dermatophagoides pteronyssinus* (DP), cat, dog, horse, feathers, ragweed, plantain, timothy grass, rye grass, *Aspergillus fumigatus* and *Alternaria tenuis*. All allergens were supplied by Hollister-Stier, Miles Inc.. In central Australia, a modified regime was used that included golden acacia, bottle brush tree, eucalyptus, melaleuca and Australian pine but did not contain dog, horse, feathers, ragweed, timothy grass or *Aspergillus fumigatus*. This modification was made because it was proposed these allergens were more appropriate for desert vegetation.

6.4.3 Bronchial Hyperresponsiveness

Forced expiratory volume in one second (FEV_1) and the forced vital capacity (FVC) were measured using Mijnhardt VRS 2000 (Mijnhardt B.V., Bunnik, Holland) dry rolling seal spirometers. The spirometers were connected to IBM compatible laptop computers running Scientific and Medical data acquisition software (S&M Instrument Company Inc., Doylestown, Pennsylvania, USA). Baseline lung function was measured while standing without a noseclip. Translators were available to assist with the forced expiratory manoeuvre instructions and subjects could see the tracing as they blew. Forced expiratory manoeuvres were repeated until they were reproducible to within 100 mls and the highest values were recorded. If the FEV_1/FVC was <60% a bronchodilator response test was performed instead (see below). Challenge tests were not performed if subjects looked too frail (eg needed assistance to stand and walk) to complete the test.

Histamine challenge tests were performed using the method of Yan *et al* (30). Once a reproducible baseline FEV_1 and FVC had been recorded the subject was taught to inhale from a De Vilbiss No. 40 glass nebuliser containing normal saline. Having mastered the technique for inhalation, a post-saline FEV_1 and FVC was recorded. The subject then inhaled increasing doses of histamine from four stock solutions. FEV_1 was recorded at 60 second intervals between the doses of histamine. The challenge was stopped if the FEV_1 fell by 20% or more or when the highest dose of histamine had been administered. The doses of histamine were doubled incrementally from 0.03 to a cumulative dose of 3.9 µmols. The higher dose of 7.8 micromols was not used in this study because this causes hoarseness of voice in about 10% of subjects, and may have discouraged participation.

Subjects who had an FEV_1/FVC <60% were given a bronchodilator test. Two puffs (200 micrograms) of salbutamol was administered via a Volumatic spacer and the spirometry was repeated after ten minutes. The response was expressed as the percentage increase in FEV_1. An increase of 15% or more was regarded as positive.

6.4.4 House Dust Mite Allergen Levels

In CY1 and CY2, dust was collected from houses of randomly selected of subjects. A two square metre area of floor or mattress was vacuumed with a hand held cleaner and the dust collected in a small bag. In central Australia people were uncomfortable with the proposal to collect dust from their houses and beds. A blanket exchange scheme was developed whereby the field team

offered a new blanket in exchange for one in current use. This program proved very popular, and allowed us to collect adequate samples of dust by vacuuming. The "old" blankets were subsequently washed and left in the clinics for community use.

In the laboratory the dust samples were sieved through a s425 stainless steel mesh by Dr Euen Tovey at Sydney University, and 0.2 gm aliquots of fine dust were examined microscopically for *Dermatophagoides* mites. Mite allergen was measured as *Der p*1 allergen by Eliza. The result was expressed as micrograms of allergen/gram of fine dust.

6.4.5 IgE Measurement

In CY1 and CY2 each asthmatic, the subject immediately following and some selected arbitrarily, had blood collected for estimation of total IgE (and HLA DR typing). Total IgE was measured by Eliza at Sydney University. The results are reported in international units (iu).

6.4.6 HLA Analysis

Blood samples were centrifuged and the buffy coat was separated for storage and transportation in dry ice. The analysis was performed at the John Curtin School of Medical Research at the Australian National University in Canberra under the supervision of Professor Sue Serjeantson. Genetic systems that could be implicated in susceptibility to atopy and asthma were examined. The blood was analysed with HLA class II cDNA probes for DR beta, DQ alpha, DQ beta, DP alpha and DP beta using southern blotting and amplified fragment length polymorphisms (73).

6.4.7 Statistics

Dummy dichotomous variables were created to identify subjects with IgE levels greater than 200 units and 999 units, atopy, asthma and foreign alleles. Sample statistics were used to calculate confidence intervals for the summary figures in the prevalence data. Chi-square tests were performed to assess the regional variation in prevalence data and Chi-square trend tests were performed to assess changes with age. Unless otherwise stated, significance tests were regarded as positive if the probability was less than 0.05. Chance adjusted Kappa scores were used to assess the inter-observer agreement regarding rhonchi. Odds ratios were used to assess the significance and magnitude of the relationships between recent wheeze and rhonchi, the presence of elevated IgE levels and asthma/atopy, and the presence of non-Aboriginal HLA alleles and asthma/atopy. Odds ratios were calculated using

standard techniques (74). The sensitivity and specificity of rhonchi for recent wheeze was assessed using the method reviewed by Sackett *et al* (75).

Logistic regression was used to calculate adjusted odds ratios for BHR and asthma (coded 0/1: 0=not present, 1=present) using proc catmod on SAS®. Age (treated as continuous), gender (coded 0/1), smoking status (coded 0/1) and atopy (to each allergen (coded 0/1)) and dummy variables for each community were initially included in the model. The least significant variable was removed sequentially. The significance of the models was assessed using maximum likelihood.

6.4.8 Comparison with Non-Aboriginal Data

During the time of this study data were collected from non-Aboriginal Australian children using similar methods (38). These data and data collected by the same epidemiological unit from adults in Busselton, Western Australia (46) were used for comparison.

6.5 Results

6.5.1 Ever Wheeze, Recent Wheeze and Rhonchi

There was significant regional variation in "ever wheeze" (Chi-square $p < 0.05$) with the prevalence ranging from 7.9% of 5-84 year olds in CA3 to 15.3% in CA4 (see table 6.2). The prevalence of "ever wheeze" significantly increased with age (Chi-square trend $p < 0.001$).

There was significant regional variation in the prevalence of "recent wheeze" (Chi-square $p < 0.001$) with the prevalence ranging from 3.1% of 5-84 year olds in CA3 to 12.4% in CA4 (see table 6.2). The prevalence of "recent wheeze" significantly increased with age (Chi-square trend $p < 0.001$).

There was significant regional variation in the prevalence of rhonchi (Chi-square $p < 0.001$) with the prevalence ranging from 2% of 5-84 year olds in CA3 to 9.2% in CA4 (see table 6.2). The prevalence of rhonchi did not significantly increase with age.

The author and another respiratory physician, working independently, examined 235 subjects for rhonchi. The chance adjusted Kappa score for the level of agreement was 0.47 (95% CI 0.28-0.69).

Table 6.3 shows a cross-tabulation of the recent wheeze and rhonchi findings using the combined data from all communities. The odds ratio for the

association was 7.6 (95% CI 4.4-13.2). Table 6.3 also shows that rhonchi predicted recent wheeze with a sensitivity of 0.24 and a specificity of 0.96.

6.5.2 Bronchial Hyperresponsiveness

Table 6.4 shows the prevalence of BHR and current asthma by age and community. The table is stratified so that the findings in the entire population (5-84 years), adults (20-84 years) or children aged 5-7, 8-12 and 13-19 years can be distinguished.

Most 5-7 year old children were unable to perform spirometry to ATS criteria and so did not receive a histamine challenge test. Over the entire age range a total of 85 histamine challenges were technically unsatisfactory (see table 6.4). The highest proportion of unsatisfactory tests occurred in CY2 (18.5%).

There was significant regional variation in the prevalence of BHR (Chi-square $p < 0.05$) with the prevalence ranging from 2.2% of 5-84 year olds in CY2 to 7.5% in CA4 (see table 6.5). The prevalence of BHR significantly increased with age (Chi-square trend $P < 0.05$).

Multivariate analyses revealed that feline allergy, house dust mite allergy (DF or DP) and smoking were significant risk factors for BHR. The adjusted (for each variable in the model) odds ratio for BHR associated with feline allergy was 1.7 (95% CI 1.01-2.30), HDM allergy was 1.6 (95% CI 1.2-2.2), and smoking was 1.4 (94% CI 1.1-1.9). Age was not significantly associated with BHR when it was adjusted for atopy.

6.5.3 Asthma

There was significant regional variation in the prevalence of asthma (Chi-square $p < 0.05$) with the prevalence ranging from 0% of 5-84 year olds in CY2 to 3.4% in CA4 (see tables 6.4 and 6.5). Chi-square trend testing did not show a significant rise in the prevalence of asthma with age but the 95% confidence intervals around the estimates for the prevalence in 8-12 year olds and 20-84 year olds did suggest that the prevalence rose significantly with age (see table 6.5).

The mean age of the 19 asthmatics was 43 years and the only one under the age of 20, was aged 11 years. Nine were males and ten were females. Seven of the asthmatics were current smokers, seven had rhonchi and eight had a loose cough. Twelve of the asthmatics (63%) were atopic to one or more allergens, with eight being atopic to DF and seven atopic to DP. Four were atopic to dust,

six to cockroaches, one plantain and one to rye grass. Three asthmatics were allergic to cats.

The multivariate analysis with variables for age, gender, community, smoking status and each allergen revealed that the only significant predictor of asthma was allergy to cat. The odds ratio for asthma in subjects allergic to cats (controlling for house dust mite, *Alternaria* and rye grass allergy) was 2.5 (95% CI 1.3 - 5.0).

6.5.4 Atopy

There was significant regional variation in the prevalence of atopy (Chi-square $p < 0.001$) with the prevalence ranging from 21% of 5-84 year olds in CA3 to 34% in CA4 (see table 6.5). Figure 6.1 shows that the prevalence of atopy increased significantly with age (Chi-square trend $p< 0.001$). Figure 6.2 shows the prevalence of atopy to the five most frequently positive allergens (DF, DP, cockroach, dust, cat) by age. Appendices 1-5 contain a detailed analysis of atopy to all the allergens by age and community.

6.5.5 House Dust Mite Sensitivity and *Der p*1 Allergen Load

Dust samples were collected from 35 houses in CY1, 20 houses in CY2, 26 blankets in CA3 and 29 blankets in CA4. The geometric mean *Der p*1 level in CY1 was 11.8 µg/g/fine dust and in CY2 was 14.9 µg/g/fine dust. In CA3 and CA4 the mean *Der p*1 levels were less than 0.05µg/g/fine dust. In table 7.6 the mean *Der p*1 levels are presented with a breakdown of the prevalence of atopy to DP by age and community. There was significant regional variation in the prevalence of atopy to DP (Chi-square $p < 0.001$) with the prevalence ranging from 14% of 5-84 year olds in CA3 to 25% in CY1 and CA4.

6.5.6 Total IgE

In CY1 and CY2, 83% of 93 subjects had IgE levels >200 units (normal < 200) and 40% had levels >999 units. Further dilutions were not performed for subjects with IgE levels greater than 1200 units. The odds ratio for the association between elevations of total IgE > 200 units and skin test atopy was 1.5 (95% CI 0.5-4.4) (see table 6.7). There was no significant association between elevated IgE above 200 units and asthma (odds ratio 1.2 (95% CI 0.1-5.8). Similarly, when subjects were divided into those with IgE levels above and below 999 units, no association could be demonstrated between elevated IgE and atopy (odds ratio 1.1 95% CI 0.5-2.5) or asthma (odds ratio 1.1 95% CI 0.3-4.9).

6.5.7 HLA Assays

Seventy-one subjects from CY1 and 25 from CY2 had blood collected for HLA DR and DQ subtype analysis. Foreign alleles were significantly more prevalent ($p < .05$) in CY1 where they were present in 23 subjects (32%) vs CY2 where they were present in 2 subjects (8%). Seven different foreign alleles were identified. They were 0301, 07, 1301, 1602, 1001, 0101 and 0103. Table 6.8 shows that the odds ratio for the association between the presence of any foreign allele and atopy (any skin test positive), was 1.2 (95% CI 0.5-3.1). Table 6.9 shows that the odds ratio for the association between the presence of foreign allele and asthma was 0.94 (95% CI 0.23-4.97).

Table 6.2
Prevalence of ever wheeze, recent wheeze and rhonchi by age group (in years) and community expressed as a percentage.

	CY1	CY2	CA3	CA4	All	95% CI
Number						
5-7	51	17	28	23	119	-
8-12	95	28	58	31	212	-
13-19	54	16	86	50	206	-
20-84	278	102	186	149	715	-
5-84	478	163	358	253	1252	-
Ever wheeze†						
5-7	8.0	0	0	4.8	4.4	0.7-8.0
8-12	2.2	0	1.7	3.3	1.9	0.1-3.7
13-19	11.3	18.8	10.6	12.0	11.8	7.4-16.2
20-84	19.9	14.3	9.7	20.9	16.6#	13.8-19.3
5-84	14.2	11.0	7.9	15.3	12.2*	10.4-14.1
Recent wheeze†						
5-7	6.0	0	0	4.8	3.5	0.2-6.8
8-12	2.2	0	1.7	3.3	1.9	0.1-3.7
13-19	7.5	0	4.7	8.0	5.9	2.7-9.1
20-84	15.4	5.1	3.2	16.9	11.1#	8.8-13.4
5-84	10.9	3.2	3.1	12.4	8.0**	6.5-9.5
Rhonchi†						
5-7	8.3	0	0	4.5	4.4	0.7-8.1
8-12	9.7	0	0	13.3	6.3	3.0-9.6
13-19	7.5	6.3	2.3	6.0	4.9	1.9-7.8
20-84	5.5	5.0	2.7	10.1	5.6¤	3.9-7.3
5-84	6.8	3.7	2.0	9.2	5.5**	4.2-6.7

Number... Number in each age group
All..... Prevalence (or numbers) when data from all communities are combined
95% CI.... Confidence interval around prevalence estimate for all
* ..... Chi-square for the difference between communities $p < 0.05$
** ..... Chi-square for the difference between communities $p < 0.001$
# ..... Chi-square trend for the significance of the increase with age $p < 0.001$
¤ ..... Chi-square trend for the significance of the increase with age not significant
† ..... Prevalence of missing values was not greater than 2%

Table 6.3

Relationship between recent wheeze and rhonchi in 5-84 year olds.

	Recent Wheeze	
	No	Yes
Rhonchi		
No	1095	74
Yes	45	23

Odds ratio = 7.56 (95% CI 4.4-13.2)

Sensitivity of rhonchi for recent wheeze is 0.24

Specificity of rhonchi for recent wheeze is 0.96

Missing values = 15

Table 6.4
Bronchial hyperresponsiveness and current asthma by age.

	N [1]	H [2]	U [3]	(%) [4]	TS [5]	BHR [6]	(%) [7]	CA [8]	(%) [9]
5-84*									
CY1	478	390	22	(5.6)	368	22	(5.9)	10	(2.7)
CY2	163	113	21	(18.5)	92	2	(2.2)	0	(0)
CA3	358	310	27	(8.7)	283	7	(2.5)	3	(1)
CA4	253	188	15	(7.9)	173	13	(7.5)	6	(3.4)
All	**1252**	**1001**	**85**	**(8.5)**	**916**	**44**	**(4.8)**	**19**	**(2)**
5-7*									
CY1	51	2	1	(50)	1	0	(0)	0	(0)
CY2	17	0	0	(0)	0	0	(0)	0	(0)
CA3	28	9	1	(11)	8	0	(0)	0	(0)
CA4	23	4	1	(25)	3	0	(0)	0	(0)
All	**119**	**15**	**3**	**(20)**	**12**	**0**	**(0)**	**0**	**(0)**
8-12*									
CY1	95	89	5	(5.6)	84	1	(1.2)	0	(0)
CY2	28	24	3	(12.5)	21	1	(4.7)	0	(0)
CA3	58	54	3	(5.5)	51	0	(0)	0	(0)
CA4	31	29	0	(0)	29	1	(3.4)	1	(3.4)
All	**212**	**196**	**11**	**(5.6)**	**185**	**3**	**(1.6)**	**1**	**(0.5)**
13-19*									
CY1	54	51	1	(1.9)	50	1	(2)	0	(0)
CY2	16	14	2	(14.3)	12	0	(0)	0	(0)
CA3	86	84	2	(2.4)	82	0	(0)	0	(0)
CA4	50	43	6	(13.9)	37	0	(0)	0	(0)
All	**206**	**192**	**11**	**(5.7)**	**181**	**1**	**(0.5)**	**0**	**(0)**
20-84*									
CY1	278	248	15	(6)	233	20	(8.6)	10	(4.3)
CY2	102	75	16	(21)	59	1	(1.7)	0	(0)
CA3	186	163	21	(12.9)	142	7	(4.9)	3	(2.1)
CA4	149	112	8	(7.1)	104	12	(11.5)	5	(4.8)
All	**715**	**598**	**60**	**(10)**	**538**	**40**	**(7.4)**	**18**	**(3.3)**

1 Number of participants
2 Number of histamine challenges (HCs)
3 Number of technically unsatisfactory HCs
4 % technically unsatisfactory HCs
5 Number technically satisfactory HCs
6 Number with BHR (positive HC)
7 % of satisfactory tests with BHR
8 Number with current asthma
9 % with current asthma (BHR and RW)
* Age in Years

Table 6.5
Prevalence of BHR, asthma (BHR plus recent wheeze) and atopy by age group and community expressed as a percentage of those tested.

	CY1	CY2	CA3	CA4	All	95% CI
Number						
5-7	51	17	28	23	119	-
8-12	95	28	58	31	212	-
13-19	54	16	86	50	206	-
20-84	278	102	186	149	715	-
5-84	478	163	358	253	1252	-
BHR						
5-7	0	0	0	0	0	0-2.4
8-12	1.2	4.7	0	3.4	1.6	0-3.3
13-19	2.0	0.0	0	0	0.5	0-1.5
20-84	8.6	1.7	4.9	11.5	7.4Ÿ	5.4-9.3
5-84	5.9	2.2	2.5	7.5	4.8*	3.6-6.0
Asthma						
5-7	0	0	0	0	0	0-2.4
8-12	0	0	0	3.4	0.5	0-1.5
13-19	0	0	0	0	0	0-1.4
20-84	4.3	0	2.1	4.8	3.3¤	1.9-4.6
5-84	2.7	0	1.0	3.4	2.0*	1.2-2.8
Atopy¥†						
5-7	14	0	7	26	13	6.9-19.0
8-12	24	18	14	29	21	15.5-26.4
13-19	28	31	28	36	30	23.7-36.2
20-84	40	43	23	36	35#	31.0-38.0
5-84	33	33	21	34	30**	27.0-32.0

Number.... Number in each age group
All..... Prevalence (or numbers) when data from all communities are combined
95% CI..... Confidence interval around prevalence estimate for all
¥ ..... Atopy to one or more allergens
† ..... Missing values = 8
* ..... Chi square for the difference between communities $p < 0.05$
** ..... Chi-square for the difference between communities $p < 0.001$
Ÿ ..... Chi-square trend for the significance of the increase with age $p < 0.05$
..... Chi-square trend for the significance of the increase with age $p < 0.001$
¤ ..... Chi-square trend for the significance of the increase with age not significant

Table 6.6

Geometric mean *Der p1* levels and prevalence of atopy (percent) to DP by age group (in years) and community.

	CY1	CY2	CA3	CA4	All	95% CI
Mean *Der p1**	11.8	14.9	< 0.05	< 0.05		
Age						
5-7	10	0	4	22	10	4.6-15.4
8-12	16	18	10	16	15	10.2-19.8
13-19	24	25	17	24	21	15.4-26.6
20-84	31	30	16	27	26#	22.8-29.2
5-84	25	24	14	25	22**	19.7-24.2

All..... Prevalence when data from all communities are combined
95% CI..... Confidence interval around prevalence estimate for all
* ..... µg/gm of fine dust
**..... Chi-square for the difference between communities $p < 0.001$
#..... Chi-square trend for the significance of the increase with age $p < 0.001$

Table 6.7

Relationship between elevated IgE and atopy.

	Atopy	
	Yes	No
IgE>200 iu	32	43
IgE<200 iu	5	10

Odds ratio for the association between atopy and IgE > 200 units 1.5 (95% CI 0.5-4.4)

N=90

Missing values = 3

Table 6.8

Relationship between non-Aboriginal HLA DR and DQ alleles and atopy.

	Atopy	
	Yes	No
Foreign Alleles Yes	11	13
Foreign Alleles No	28	41

Odds ratio for the association between Atopy and foreign alleles 1.2 (95% CI. 0.5-3.1)

N= 93

Missing values = 3

Table 6.9

Relationship between non-Aboriginal HLA DR and DQ alleles and asthma.

	Asthma	
	Yes	No
Foreign Alleles Yes	2	23
Foreign Alleles No	6	65

Odds Ratio= 0.94 (95% CI 0.23-4.97)

N=96

Figure 6.1

Regional variation in atopy by age.

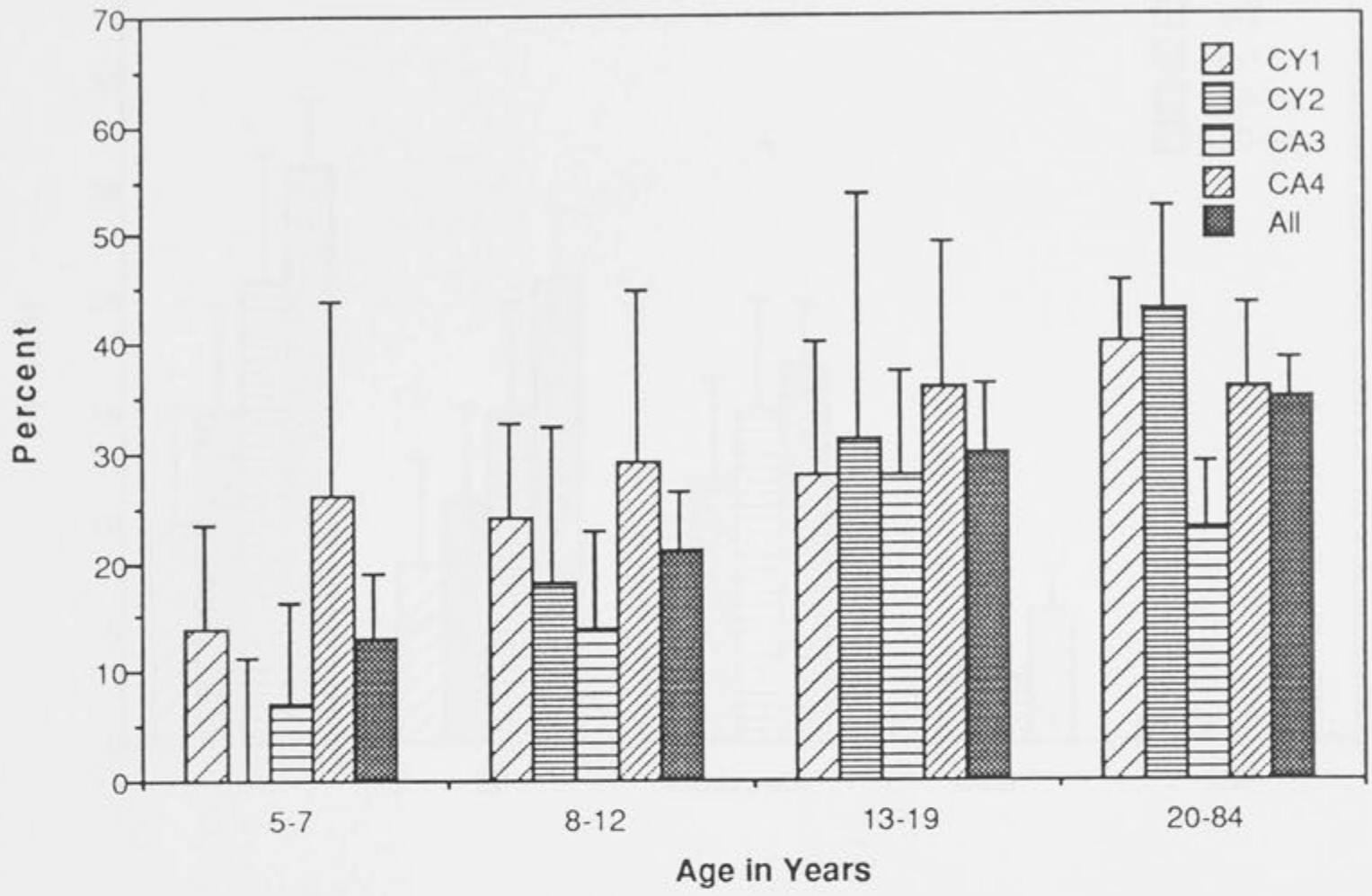


Figure 6.2

Atopy to DP, DF, cockroach, dust, cat, by age group (in years).

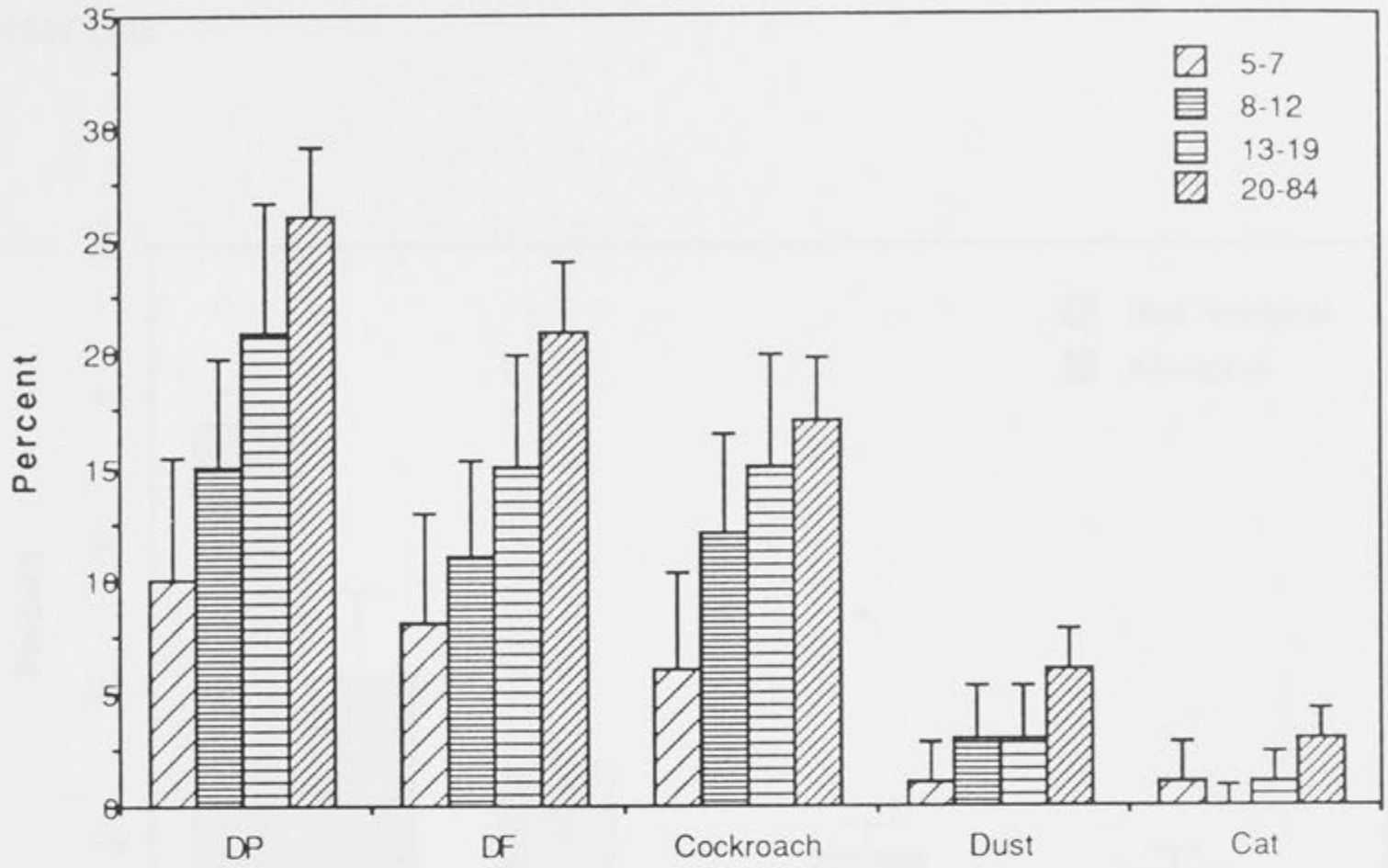


Figure 6.3

Atopy, recent wheeze, BHR and asthma in 8-12 year old Aboriginal children and 8-10 year old non-Aboriginal children. Non-Aboriginal data from Wagga Wagga, NSW (38).

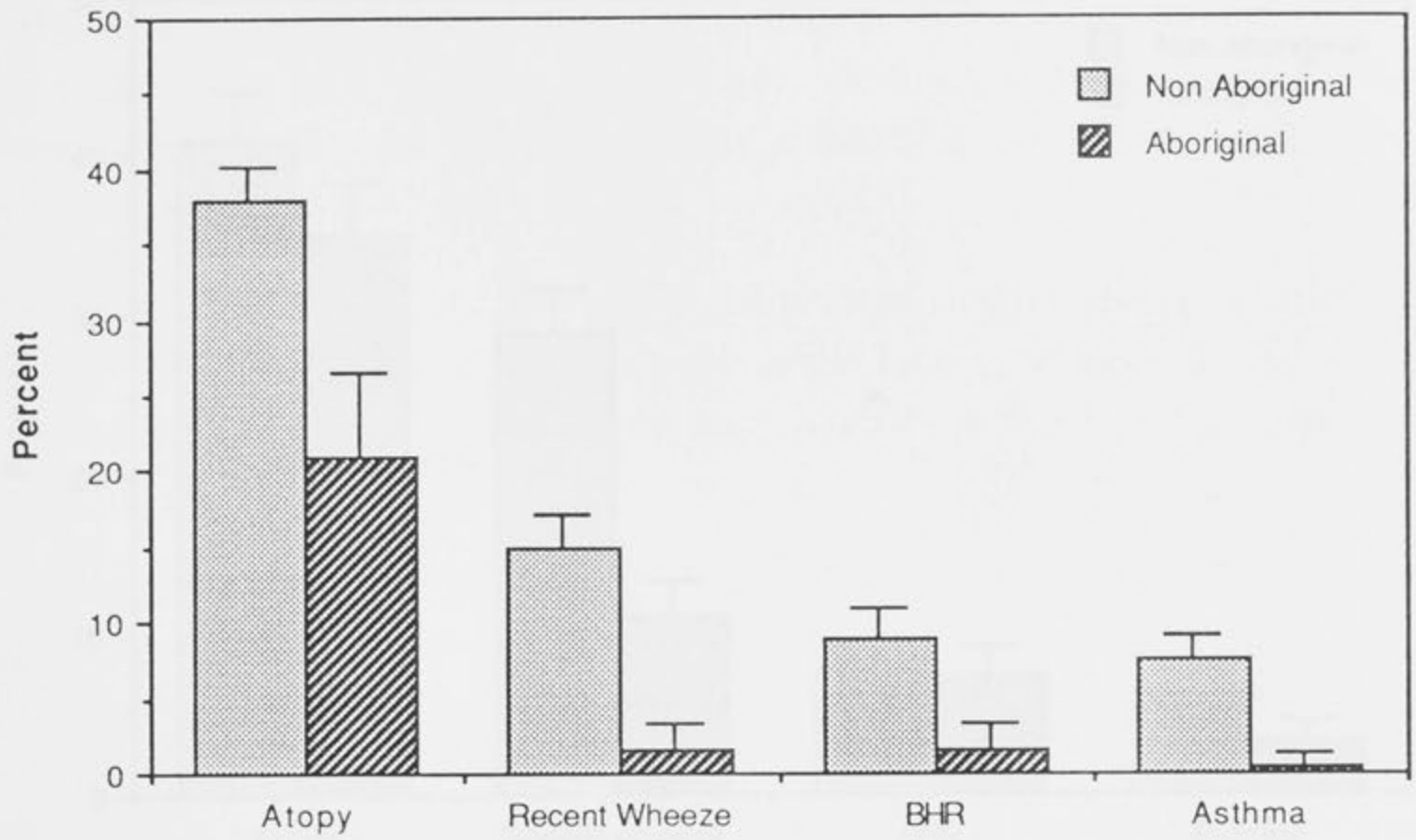
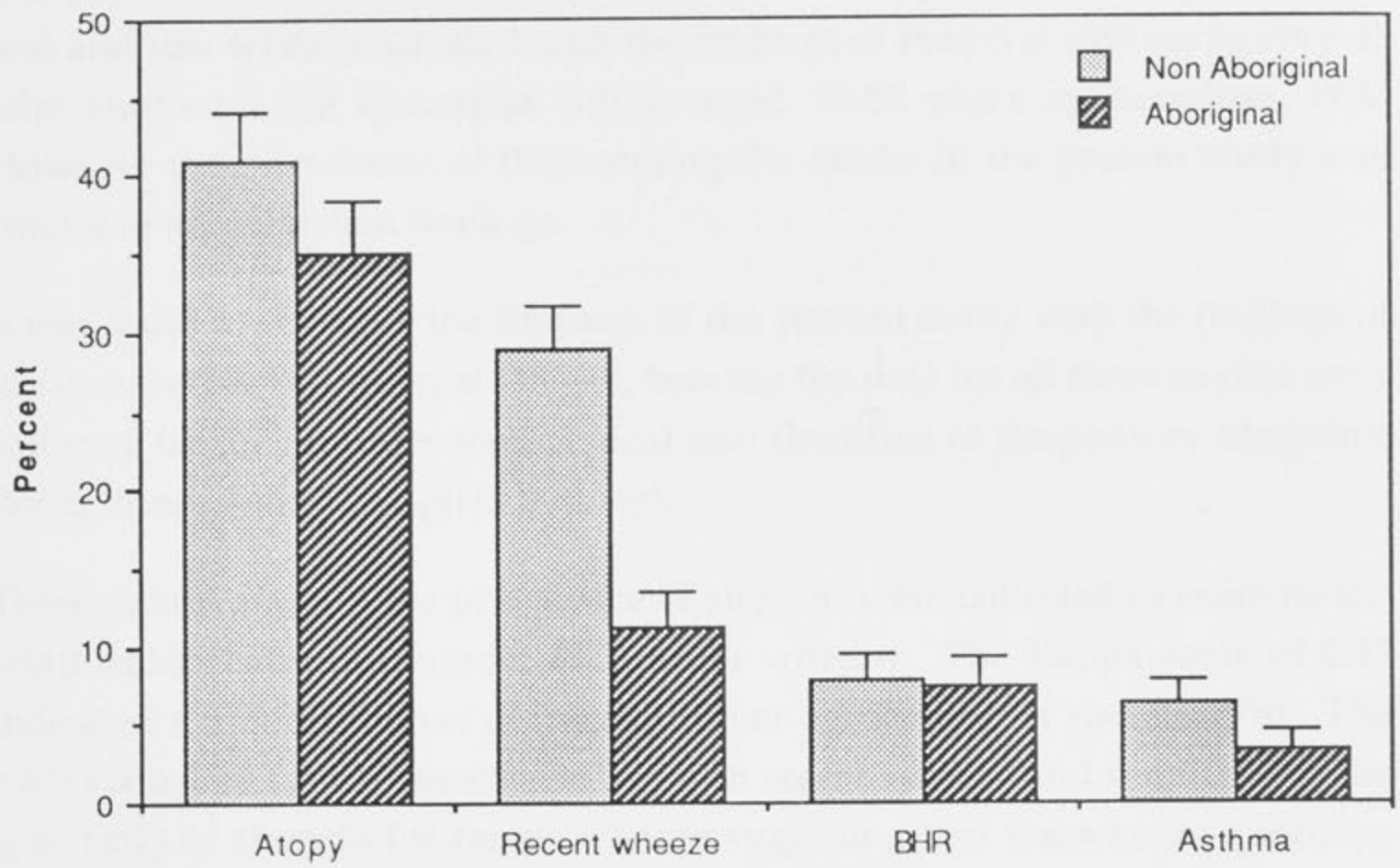


Figure 6.4

Atopy, recent wheeze, BHR and asthma in Aboriginal and non-Aboriginal adults in Australia. Non-Aboriginal data from Busselton, WA (46).



6.6 Discussion

The prevalence of recent wheeze, BHR, atopy and asthma in the 8-12 year old Aboriginal children was low when contrasted with the findings of Peat et al (38)(see figure 6.3) who studied 1,668 non-Aboriginal children aged 8-10 years in Wagga Wagga, NSW.

The prevalence of recent wheeze, asthma and atopy in the Aboriginal adults was also low when contrasted with the findings of Peat *et al* (46)(see figure 6.4) who studied 1,028 Caucasian adults aged 18-55 years in Busselton, WA. However the prevalence of BHR among the adults in the present study was similar to the Busselton findings.

It was valid to compare the findings of the present study with the findings of the two studies by Peat *et al* (38, 46), because the data for all three studies were collected by the same epidemiological unit (Institute of Respiratory Medicine, Royal Prince Alfred Hospital, Sydney).

These data regarding the prevalence of rhonchi were collected to examine the relationship between rhonchi and recent wheeze. The Kappa score of 0.47 indicated a moderate level of inter-observer agreement for rhonchi (76). The odds ratio (7.6) for the association between recent wheeze and rhonchi, and the specificity of rhonchi for recent wheeze suggested that the wheeze questions were valid and therefore it was unlikely that the low prevalence of recent wheeze was an artefact of the questionnaire design or difficulty with translation. The low sensitivity of rhonchi for detecting recent wheeze was expected because rhonchi are evanescent with disease activity.

The present study shows that asthma in rural Aborigines is predominantly a disease of adults. This pattern has been described by Dowse *et al* in PNG (77), Warrell *et al* in Nigeria (78), and in other tropical environments. Despite several hypotheses regarding the importance of genetic, immunological and environmental factors the reasons for the low prevalence of asthma in these areas remain unknown (79).

Although the prevalence of asthma was generally low there was significant regional variation (prevalence high in CY1 and CA4 and low in CY2 and CA3) (see table 6.5). The participation rate from CA3 was high, so it is unlikely sampling bias explains the low prevalence of asthma there. Although the participation rate was relatively poor in CY2, Dr Thompson has never seen a person with asthma there (despite ten years of regular visits to conduct chest

clinics) so it is unlikely that poor participation explains the low prevalence in CY2. The two communities with a relatively high prevalence of asthma had starkly contrasting living conditions and climates. Although the cause of regional variation in asthma prevalence is unknown some possible explanations are discussed below.

The high mite allergen levels in Cape York and the low levels in central Australia were predicted from their climates (67). Surprisingly the prevalence of HDM atopy in CA4 was similar to CY1 and CY2 despite CA4 having essentially undetectable levels of *Der p*1. It has been shown that cross-antigenicity exists between *Sarcoptes scabiei* (scabies) and *Dermatophagoides pteronyssinus* (80). Scabies is common in Aboriginal communities (54), so perhaps cross-antigenicity may explain the high prevalence of atopy to DP in CA4. However scabies infection also occurs in CA3 so it is not clear why there was a lower prevalence of sensitivity to HDM in this community. This putative cross-reaction is important because it could have reduced the power of the study to detect an association between HDM atopy and BHR or asthma (see below).

The contribution of HDM allergen exposure to the development of atopy, BHR and asthma was unclear in this study. Mean levels of *Der p*1 in CY1 and CY2 were high enough to cause sensitisation and wheezing (66), and HDM atopy was a risk factor for BHR. However, the prevalence of asthma atopy and BHR among children was low. This finding suggested that Aboriginal children are protected from the pulmonary effects of high *Der p*1 exposure. Aboriginal children also appear to be "protected" from developing sensitivity to other allergens (see below). One possible mechanism and some conclusions regarding the importance of this observation are also discussed below.

The distribution of the non-Aboriginal HLA DR and DQ alleles supported the hypothesis that the people of CY1 have experienced relatively more genetic mixing than those in CY2. However, there was no evidence that asthma or atopy in CY1 were linked to the foreign genes. These findings suggest that environmental, rather than genetic factors, should be invoked to explain the higher prevalence of asthma in CY1 compared with CY2. As the people in both these communities lived in houses with similar levels of HDM allergen exposure it is unlikely differences in exposure to *Der p*1 account for the findings. Other possible environmental factors therefore need to be considered.

It is likely that the high levels of total IgE found in CY1 and CY2 reflected a high prevalence of intestinal parasite infection with *Trichuris trichuria* and *Ascaris lumbricoides* (68, 81). This putative association with parasite infestation may explain why there was no relationship between elevated IgE levels and the prevalence of atopy or asthma. Presumably parasite-specific IgE "swamped" any elevations of IgE associated with allergy to aero-allergens. These data are consistent with the observation of Carswell *et al* (82) who showed that parasite infestation did not protect Tanzanian children from developing asthma.

The finding that BHR was significantly associated with atopy to cats and house dust mites (DF or DP) (see 6.5.2) was consistent with the findings of Peat *et al* (46). They found that the adjusted odds ratio for BHR in Caucasians was 5.5 for house dust mite allergy and 3.1 for feline allergy. The finding was important because the apparently low prevalence of BHR in Aboriginal children may be secondary to the low prevalence of atopy (see figure 6.3). In adults the association between BHR and atopy was important because the regional variation in the prevalence of atopy may explain the regional variation in BHR (and asthma) (see table 6.5). A weak association between smoking and BHR is well recognised (45) but it is unlikely that regional variation in the prevalence of smoking is significantly affecting the prevalence of BHR because in CY2, where smoking was most prevalent, the prevalence of BHR was low.

In the present study the low prevalence of atopy in children (see figure 6.3) was unlikely to have been due to the use of inappropriate allergens because the prevalence of adult atopy to the same allergens was significantly higher and similar to that found in non-Aboriginal Australians (see figure 6.4). Therefore Aboriginal Australians appear to have a slower rate of atopy acquisition than non-Aboriginal Australians. Because atopy has been associated with BHR in many studies (including the present study) this slow rate of atopy acquisition may be an important factor explaining the generally low prevalence of asthma among Aborigines.

One possible cause for the slow acquisition of atopy in children is chronic, profuse, purulent nasal discharge. Perhaps chronic nasal discharge is attenuating the presentation of aero-allergens to the naso-bronchial mucosa thus retarding sensitisation.

The finding that feline allergy was a risk factor was similar to the findings of Peat *et al* (46). Cats live in each of the four study communities. The multivariate analysis performed to explore the associations with asthma was

necessarily hindered by the small numbers (19 asthmatics). Therefore the power to detect associations with asthma was limited. Peat *et al* (46) also demonstrated an association with HDM allergy that was not present in the Aboriginal asthmatics. This lack of association between HDM allergy and asthma in the present study was unexpected because there was an association between HDM allergy and BHR. It is possible that a cross-reaction between *Sarcoptes scabiei* and *Dermatophagoides pteronyssinus* (see above) reduced the strength of any relationship between house dust mite allergy and asthma, and that this, combined with the low power of the study resulted in the negative finding. A large case-controlled study of Aboriginal asthmatics could clarify whether HDM allergy is an important risk factor for asthma.

It is not clear why the findings in the present study regarding the prevalence of atopy and BHR do not concur with the findings of Clarke *et al* (see chapter 2) (50). It is possible that the differences seen with the comparison between the two studies are a result of confounding by age or differences in the criteria used to assess atopy and BHR.

Discussions with Aboriginal medical service staff in Melbourne and Sydney suggest that asthma occurs commonly in urban Aboriginal children. In other cultures changes in the prevalence of asthma have been found in association with migration to new environments. For example Waite *et al* found that the prevalence of clinically diagnosed asthma amongst Tokelauen children aged 0-14 was 11.0% in Tokelau and 25.3% in New Zealand (Clinical Allergy 1980;10:71-75). In addition Waite *et al* found evidence to suggest that atopic eczema and allergic rhinitis were more prevalent amongst the Tokelauen children living in New Zealand. The reasons for these changes with migration are unknown. A study of asthma among urban Aboriginal children is needed to determine the prevalence of asthma and explore the associations with atopy. Urban Aboriginal children may develop atopy at an earlier age than their rural counterparts and as a consequence have a higher prevalence of BHR and asthma.

6.7 Conclusion

- The prevalence of asthma in adults was low in comparison with that found among non-Aboriginal Australians and in children it was almost non-existent.
- The only significant risk factor for asthma was allergy to cats.
- The prevalence of asthma in Cape York children was low despite exposure to high levels of *Der p*1 allergen.
- The prevalence of BHR among adults was similar to that found in non-Aboriginal Australians but in children the prevalence was low.
- The risk factors for BHR were HDM allergy, feline allergy and cigarette smoking.
- There was significant regional variation in the prevalence of atopy and asthma.
- The prevalence of atopy in adults was similar to that found among non-Aboriginal Australian adults but in children it was significantly lower than that found among non-Aboriginal children of the same age. Aboriginal children may have delayed atopy acquisition.
- There was no evidence that asthma or atopy in CY1 and CY2 were associated or linked with non-Aboriginal HLA DR and DQ alleles.
- IgE levels were not helpful in identifying those at risk for BHR or asthma.
- The low prevalence of asthma in Aborigines is unexplained but the regional variation is possibly due to environmental factors that affect the acquisition of atopy.

CHAPTER 7

Normal Lung Function

7.1 Introduction

Little is known about normal lung function in Aborigines. Studies by Chandler *et al* (26) and Watson *et al* (52) showed that FEV_1 and FVC in Aboriginal children were approximately 25% lower than those predicted for Caucasian children. However children with signs or symptoms of lung disease were not excluded from the reference populations in these studies so the equations published by these authors may not predict "normal" lung function (48).

Thompson *et al* have published equations for normal lung function in Aboriginal adults but further studies are required to confirm the findings (1). They measured FEV_1 and FVC in 229 Aboriginal adults and found them to be 25% lower than values predicted by equations developed for Caucasians. They also found that age related decline in lung function appeared higher than that found in Caucasians, and most surprisingly, that smokers appeared to have less age related decline in lung function than non-smokers (see chapter 2).

It is recognised that there are racial variations in lung function (83-85). Lung function equations derived from Caucasian populations usually over-estimate values in black subjects by 7-20%. Some of these differences may be secondary to anthropometric differences between races because when compared with Caucasians, some indigenous peoples have relatively long legs (or relatively short chests) per unit of standing height (86). Standardisation using sitting height rather than standing height has been shown to reduce but not eliminate

racial differences in ventilatory function (48). Also environmental factors have been postulated to explain racial differences in lung function (48).

No unified approach has been adopted for modelling pulmonary function using cross-sectional data (87). Multiple linear regression using age and height as the main independent variables has been the method adopted by most investigators (48, 88). However linear models cannot describe adequately the changes in FEV_1 and FVC that occur during the transition from lung growth in the late teenage years, to the plateau in the early twenties and then the subsequent decline with age (88). Recently spline smoothing has been used to portray the growth and decay of FEV_1 and FVC with age. Unfortunately this technique does not yield reference values (89).

7.2 Aims

The aim here was:

- To develop equations to predict FEV_1 and FVC in children and adults;
- To estimate the rate of decline of lung function with age for adults;
- To examine the influence of smoking on lung function in asymptomatic smokers to determine whether they have less age related decline than non-smokers.

7.3 Methods

A reference population was identified by excluding those subjects with known lung disease, chronic cough, recent wheeze, loose cough, crepitations, rhonchi and BHR. Subjects with BHR were excluded because BHR may be associated with abnormalities of ventilatory function (90).

7.3.1 Measurement of FEV_1 and FVC

FEV_1 and FVC were measured using Mijnhardt VRS 2000 (Mijnhardt B.V., Bunnik, Holland) dry rolling seal spirometers while subjects were standing (without noseclips). The tests were performed using ATS criteria (91) by technicians from Professor Woolcock's epidemiological unit (see 6.4.3). The spirometers were calibrated twice daily and all values were converted to BTPS.

7.3.2 Height

Standing height was recorded in centimetres with footwear removed. Sitting height was measured using a bench and a vertically mounted tape measure. The base of the tape measure was set level with the top of the bench.

7.3.3 Analysis

Visual inspection of plots of FEV_1 and FVC with age confirmed that spirometric growth was complete by the age of 19 years. Therefore these data were divided into 7-19 year olds and 20-80 year olds and analysed separately. Smoking and gender were coded as dichotomous variables. Standing and sitting height (measured in cm) and age in years were treated as continuous variables.

Multiple linear regression was utilised to develop equations to predict FEV_1 and FVC using SAS® (92). Gender, smoking, age, standing height and sitting height were used as the independent variables. The models were fitted using the least squares technique. Non-linear effects of age and height were examined by using height squared, age squared, $\log_e$ height and $\log_e$ age. Interactions between gender, smoking, age and height were also examined. Statistical significance was assessed at the 0.05 level. Plots of residual values against predicted values were examined for all models to assess trends in the unexplained variance. The 95% percent confidence intervals for the modelled values of FEV_1 and FVC were calculated by taking account of the point variance of the residuals as well as the variance of the parameter estimate.

7.4 Results

7.4.1 Data Base Summary

After the exclusion criteria were applied, the reference population comprised 261 subjects aged 7-19 years, and 332 subjects aged 20-80 years. Table 7.1 shows the structure of the reference population by age, gender and smoking status. The mean values of the dependent and independent variables in 7-19 year olds (plus SD and range) are detailed in Table 7.2, and Table 7.3 shows these data for 20-80 year olds. The crude correlations (Pearson R values) between the dependent and independent variables for 7-19 year olds are presented in table 7.4, and table 7.5. shows these data for 20-80 year olds.

Table 7.1

Reference population stratified by age, gender and smoking status.

	Males	Females
7-19 Year olds (N=261)		
Smokers	25	11
Non-smokers	108	117
Total	133	128
20-80 Year olds (N=332)		
Smokers	72	59
Non-smokers	58	143
Total	130	202

Table 7.2

Mean values of the dependent and independent variables and FEV_1/FVC ratio in the childhood reference population.

7-19 Year olds	Mean	SD	Minimum	Maximum
MALE				
Age	12.9	3.2	7	19
Height	153.0	16.6	119	181
Sitting height	75.2	8.3	58	91
FEV_1	2.3	0.8	0.97	4.02
FVC	2.5	0.89	1.08	4.59
Ratio FEV_1/FVC	93.1	5.3	78	100
FEMALE				
Age	12.8	3.8	7	19
Height	148.2	14.7	115	176
Sitting height	73.7	7.3	58	88
FEV_1	2.0	0.6	0.8	3.4
FVC	2.1	0.7	0.8	3.5
Ratio FEV_1/FVC	94.2	4.9	80	100

Height was measured in centimetres.

Table 7.3

Mean values of the dependent and independent variables and FEV_1/FVC ratio in the adult reference population.

20-80 Year olds	Mean	SD	Minimum	Maximum
MALE				
Age	38.1	13.8	20	80
Height	173.9	6.2	159	195
Sitting height	85.8	3.8	74	97
FEV_1	3.05	0.7	1.3	4.9
FVC	3.4	0.8	1.5	6.2
Ratio FEV_1/FVC	87.8	7.2	67	100
FEMALE				
Age	34.4	12.2	20	78
Height	160.9	6.1	149	188
Sitting height	80.1	3.7	69	91
FEV_1	2.3	0.5	0.9	3.7
FVC	2.5	0.6	1.1	4.3
Ratio FEV_1/FVC	91	6.8	66	100

Height was measured in centimetres.

Table 7.4

Correlation coefficients (Pearson R values) between the dependent and independent variables in the childhood reference population.

	Standing height	Sitting height	Age
Male non-smokers			
FEV_1	0.88***	0.87***	0.83***
FVC	0.87***	0.86***	0.84***
Male smokers			
FEV_1	0.70***	0.66**	0.22*
FVC	0.60**	0.64**	0.27*
Female non-smokers			
FEV_1	0.85***	0.84***	0.75***
FVC	0.86***	0.85***	0.76***
Female smokers			
FEV_1	0.77**	0.80**	0.33*
FVC	0.78**	0.69**	0.37*

Height was measured in centimetres.

* $p = N/S$

** $p < 0.05$

*** $p < 0.0001$

Table 7.5

Correlation coefficients (Pearson R values) between the dependent and independent variables in the adult reference population.

	Standing height	Sitting height	Age
Male non-smokers			
FEV_1	0.35**	0.34**	-0.65***
FVC	0.40**	0.32**	-0.60***
Male smokers			
FEV_1	0.53***	0.48***	-0.40**
FVC	0.56***	0.47***	-0.02*
Female non-smokers			
FEV_1	0.51***	0.58***	-0.44***
FVC	0.56***	0.62***	-0.37***
Female smokers			
FEV_1	0.34**	0.62***	-0.31**
FVC	0.41**	0.66***	-0.21*

Height was measured in centimetres.

* $p = N/S$

** $p < .05$

*** $p < 0.0001$

7.4.2 Models for FEV_1 and FVC

In children, the final models for FEV_1 and FVC (Table 7.6) contained terms for height, age, gender and the interaction of gender with age. Smoking status was omitted because it did not have a significant effect in the model. The final models for adults (see table 7.6) contained terms for height, age, gender and smoking. Adult cigarette smokers had higher predicted values of FEV_1 (by 138 mls) and FVC (by 260 mls) than non-smokers. Table 7.6 also shows the root mean square error and R^2 values associated with the models.

Two examples are provided to demonstrate use of the models:

1. To calculate FVC in a 14 year old female who is 155 cm tall:

FVC = - 3.506 + (0.03215 x ht) + (0.08453 x age) + (0.43965 x gender) - (0.05029 x gender x age)

= - 3.506 + (0.03215 x 155) + (0.08453 x 14) + (0.43965 x 1) - (0.05029 x 1 x 14)

= - 3.506 + 4.983 + 1.183 + 0.4396 - 0.70406

= 2.40 litres

2. To calculate FEV_1 in a 40 year old male smoker who is 175 cm tall:

FEV1 = - 3.469 + (0.04169 x ht) - (0.02133 x age) - (0.23036 x gender) + (0.13786 x Smoking status)

= - 3.469 + (0.04169 x 175) - (0.02133 x 40) - (0.23036 x 0) + (0.13786 x 1)

= - 3.469 + 7.29575 - 0.8532 - 0 + 0.13786

= 3.11 litres

To estimate the 95% confidence interval of any predicted value multiply the appropriate root mean square error (RMSE) value by 1.96 and add or subtract it from the predicted value. In example (1) above the confidence interval is as follows:

95% CI = $2.40 \pm 0.373 \times 1.96$

= 2.40 ± 0.731

= (1.67 - 3.13)

Table 7.6

Models for FEV_1 and FVC in 7-19 year old and 20-80 year old Aborigines.

	7-19 Year olds		20-80 Year olds	
	FEV_1	FVC	FEV_1	FVC
Intercept	-3.354	-3.506	-3.469	-5.159
(SE)	0.273	0.305	0.661	0.798
Height	0.03095	0.03215	0.04169	0.05315
(SE)	0.00260	0.00290	0.00377	0.00455
Age	0.07321	0.08453	-0.02133	-0.01961
(SE)	0.01471	0.01646	0.00189	0.00228
Gender	0.40771	0.43965	-0.23036	-0.23870
(SE)	0.16092	0.17997	0.06933	0.08369
Current smoker	-	-	0.13786	0.26047
(SE)	-	-	0.05181	0.06254
Gender x age	-0.04455	-0.05029	-	-
(SE)	0.01227	0.01372	-	-
RMSE	0.33	0.37	0.42	0.51
R^2	0.80	0.79	0.62	0.62

Coding for gender: males = 0, females = 1

Coding for smoking status: non-smokers = 0, smoker = 1

Height = standing height in centimetres

Age = age in years

Gender x age = Gender times age

- indicates not applicable

The probability p of all estimates is < 0.05

SE = Standard error

RMSE = Root mean square error

R^2 = square of Pearson correlation coefficient

All spirometric values in litres BTPS

The following tables have been compiled to allow the prediction of FEV_1 and FVC in Aboriginal adults and children:

Appendix 6	FEV_1	Males	9-19 years old
Appendix 7	FVC	Males	9-19 years old
Appendix 8	FEV_1	Male non-smokers	20-70 years old
Appendix 9	FVC	Male non-smokers	20-70 years old
Appendix 10	FEV_1	Male smokers	20-70 years old
Appendix 11	FVC	Male smokers	20-70 years old
Appendix 12	FEV_1	Females	9-19 years old
Appendix 13	FVC	Females	9-19 years old
Appendix 14	FEV_1	Female non-smokers	20-70 years old
Appendix 15	FVC	Female non-smokers	20-70 years old
Appendix 16	FEV_1	Female smokers	20-70 years old
Appendix 17	FVC	Female smokers	20-70 years old

7.5 Discussion

Although 1,000 subjects completed spirometric testing, the exclusion of those with technically unsatisfactory tests, signs or symptoms of lung disease, bronchial hyperreactivity and airflow obstruction left only 593 subjects for lung function modelling (Table 7.1). The 40% reduction in numbers reflected the high prevalence of respiratory disease in these communities (see chapter 9). Nevertheless the numbers available for modelling were comparable to many studies used to develop reference values in Caucasian populations (48).

In the present study, the mean age and height of adult men and women in the reference population was similar to that in Thompson *et al's* study (1). This similarity between the two populations was important as it allowed direct comparison of predicted values at mean age and height where both sets of equations were likely to be most accurate.

The significant correlations between height, age and ventilatory capacity were expected (88). Although height and age were highly correlated (positively in children and negatively in adults), both were included as independent variables in the modelling because it is recognised that lung growth and maturation may occur independently of height (particularly in the late teenage years) (88). The finding that sitting height was no better a predictor of lung

function than standing height supports the finding of Thompson *et al* (1). This was expected as sitting and standing height are highly correlated.

The R^2 values for the models (see table 7.6) showed that height and age accounted for 80% of the variance of FEV_1 and FVC in children, and 62% of the variance in adults. These R^2 values were comparable to those of Burrows et al (88) who used exponential terms of height and age to describe lung function in 916 non-smokers aged six years and over. The R^2 values were also similar to those obtained by Thompson *et al* (1). The standard errors for the models in the current study were comparable to many other lung function reference value studies (48). Thus the models in the present study probably explained as much of the variance in FEV_1 and FVC as is possible with linear equations.

Table 7.7 and figure 7.1 show comparisons of predicted values of FEV_1 and FVC using the childhood equations from the present study, those of Chandler et al (26) and those of Watson *et al* (52). The values were calculated for children at the mean age and height of children in the present study. Both Chandler *et al's* and Watson *et al's* equations predicted values within the 95% CI of the present study models. However Chandler *et al's* equations predicted values within 100 mls of the present study whereas those of Watson *et al* did not. The close agreement between the present study models and Chandler *et al's* predictions suggested that both models were accurate in mid-childhood.

Significant differences in predicted spirometric values can occur at the "transition" from childhood to adult lung function models (88). Table 7.8 and Figure 7.2 show a range of predictions for FEV_1 and FVC at the age of 19 and 20 years (using the mean height of adult men and women in the present study). The predicted values of FEV_1 and FVC at age 19, using the childhood equations of Chandler *et al* (26), Watson *et al* (52) and the present study, are contrasted with the predicted values at age 20 using adult equations of the present study and Thompson *et al* (1). These data showed that the childhood equations from the present study (at age 19), closely predict the FEV_1 and FVC predicted by the adult equations from the present study (at age 20) (within 50 mls for males and 20 mls for females). This consistency suggests that the models developed in the present study are robust. Table 7.8 and figure 7.2 also suggest that the models of Watson *et al*, Chandler *et al*, and Thompson *et al* do not accurately predict ventilatory function in these "transition" years.

Figures 7.3 to 7.6 compare the predicted spirometric values for adult non-smokers (at the mean height of adults in the present study) using the equations

developed for Aborigines in the present study and by Thompson *et al* (1), with the corresponding values predicted for Caucasians by Morris *et al* (93). The similarity of the predicted values at approximately the mean age of adults (38 years in men and 34 years in women) between the present study and that of Thompson *et al* suggested that both sets of equations satisfactorily predicted spirometric function at this point. However the equations of Thompson *et al* appeared to over-predict ventilatory function in the early adult years and exaggerate the decline with age. The validity of the present study's predicted values at aged 20 years was enhanced by the concordance with the values predicted for 19 year olds by the present study's childhood equations (see above). The adult equations of the present study probably predict normal spirometric function in Aborigines more accurately than do Thompson *et al's* because the reference population was 50% larger, the age distribution was wider and subjects with BHR were excluded.

These data in figures 7.3 to 7.6 also support the observation of Thompson *et al* (1) that FEV_1 values in Aborigines are approximately 20% lower than those found in Caucasians (of the same standing height, age, and gender), and that FVC values are approximately 30% lower. This suggests that Aboriginal spirometric volumes are even lower than those found in black Americans, black Africans and Indians (48, 83, 84). Although anthropometric differences between Aborigines and Caucasians may partially explain the low vital capacity (and FEV_1), it is possible that compromised lung growth in childhood is responsible. Low socio-economic status has been associated with significant reductions in FVC in American children (94). It is not known if passive and active smoking during childhood, lower respiratory tract infections and other factors are affecting lung growth in Aboriginal children. In chapter nine it will be shown that petrol sniffing is associated with abnormalities of ventilatory function. Whether the small lung volumes in Aborigines are due to anthropometric effects, attenuated lung growth or other factors is unresolved.

The observation that the FVCs of subjects in the present study were 30% lower than those found in Caucasians of the same age, gender and height and that FEV_1s were only 20% lower (see above) is the most likely reason that the FEV_1/FVC ratios in the present study were high (see tables 7.2 and 7.3) in comparison with those found in Caucasians (95). This means that ratios that would be considered "normal" in Caucasians may not be normal in Aborigines. High FEV_1/FVC ratios have also been found in many other non-Caucasian races (48).

Cross-sectional studies of lung function are prone to cohort effects and tend to over-estimate the true rate of decline of lung function with age (87). It is likely that rapid changes in Aboriginal society over recent generations have resulted in significant cohort effects which in turn could have influenced pulmonary function. Figures 7.3 to 7.6 show that the predicted spirometric values from the present study and that of Morris *et al,* slowly converged with age. This suggests that the rate of decline of ventilatory function in healthy adult Aborigines is no greater than that in Caucasians. This conclusion contrasted with the findings of Thompson *et al* (1) whose equations suggest Aborigines have a greater rate of lung function decline than Caucasians.

The finding that smokers had larger predicted values for FEV_1 and FVC than non-smokers was surprising because smokers usually have lower values for spirometry than non-smokers (48). However this was partially consistent with the findings of Thompson *et al* (1) although they also found an interaction with age that suggested non-smokers had a greater rate of decline in FEV_1 and FVC than smokers. This was not the case in the present study. The findings in the present study could be explained by a health selection effect. It has been observed by Gold *et al* (5) that children previously hospitalised for respiratory illness are less likely to commence smoking than children without such a history. Gold *et al* hypothesised that this could produce a "healthy smoker effect" where children who are constitutionally less comfortable smoking choose not to take up the habit. In the present study, where the prevalence of chronic lung disease in non-smokers is high, "healthy smoker effects" could be important. Aboriginal people may be less likely to commence or persist with cigarette smoking if they have an existing ventilatory impairment. Such a health selection effect (bias) could explain why the smokers appeared to have better lung function than non-smokers. Alternatively the exclusion criteria could have removed smokers with low normal or borderline lung function from the reference population leaving asymptomatic smokers with relatively good lung function.

7.6 Conclusion

- The equations developed in the present study predict spirometric values at the mean height and age of children (and adults) that are similar to the findings of other authors.
- The childhood and adult equations from the present study predict concordant spirometric values at 19 and 20 years of age.
- These spirometric equations predict values at 19 and 20 years of age that are discordant with equations developed by previous authors.
- These spirometric equations are probably more valid than previously published equations because they were developed from a relatively large reference population which was free of symptoms or signs of lung disease and BHR.
- Cigarette smokers have higher predicted values of FEV_1 and FVC than non-smokers.
- The values predicted for FEV_1 in Aborigines by the equations developed in the present study are approximately 20% lower than those predicted for Caucasians of the same height, age and gender.
- The values predicted for FVC in Aborigines by the equations developed in the present study are approximately 30% lower than those predicted for Caucasians of the same height, age and gender.
- The FEV_1 /FVC ratio in healthy Aborigines is higher than that found in Caucasians.
- The rate of decline of FEV_1 and FVC in healthy Aborigines appears no greater than that observed in Caucasians.
- The rate of decline of FEV_1 and FVC in asymptomatic Aboriginal smokers appears no greater that found in non-smokers.
- More research is required to investigate the determinants of lung function growth and decline in Aborigines.

Table 7.7

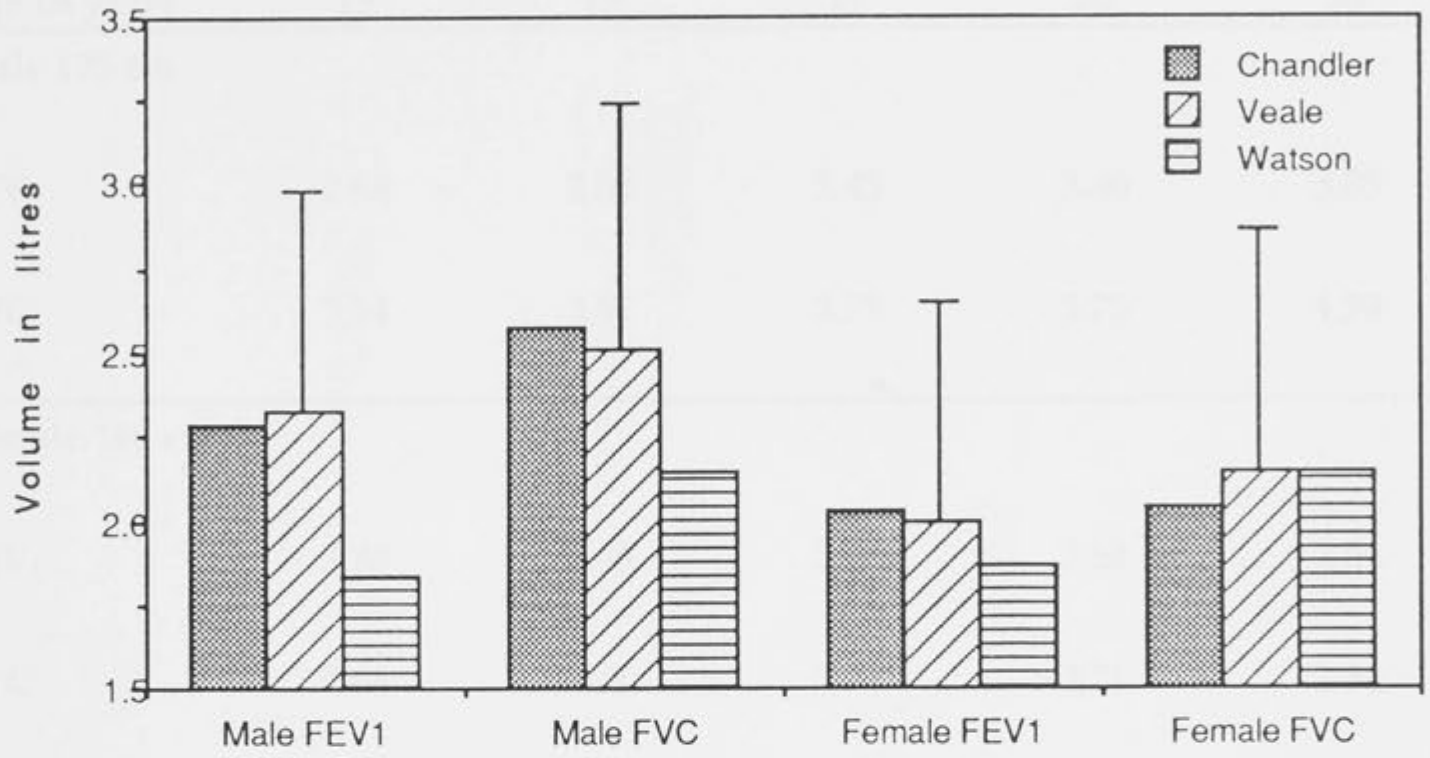
Comparison of predicted values of FEV_1 and FVC (litres) for the present study, Chandler *et al's* study (26) and Watson *et al's* study (52). Comparison is made at the mean age and height for males and females from the present study.

	Predicted	95% CI
Males (aged 13, height 153 cm)		
FEV_1		
Present study	2.33	1.68 - 2.98
Chandler *et al*	2.28	*
Watson *et al*	1.83	*
FVC		
Present study	2.51	1.78 - 3.24
Chandler *et al*	2.58	*
Watson *et al*	2.14	*
Females (aged 13, height 148 cm)		
FEV_1		
Present study	2.00	1.35 - 2.65
Chandler *et al*	2.03	*
Watson *et al*	1.87	*
FVC		
Present study	2.14	1.41 - 2.87
Chandler *et al*	2.04	*
Watson *et al*	2.14	*

* indicates standard error of estimates was not reported.

Figure 7.1

Comparison of predicted FEV_1 and FVC in 13 year old Aboriginal children using the equations of Watson *et al* (52), Chandler *et al* (26) and the present study. Comparison is made at the mean height of male (153 cm) and female (148 cm) children from the present study.



Chandler: The predicted values of FEV_1 and FVC using the paediatric equations of Chandler *et al* (26).

Veale: The predicted spirometric values using the paediatric equations from the present study.

Watson: The predicted spirometric values using the paediatric equations of Watson *et al* (52)

The T bars indicate the upper bounds of the 95% confidence interval for Veale.

FEV_1 and FVC measured in litres.

Table 7.8

Table comparing the predicted spirometric values for non-smokers, at the mean height of adults, at the "transition" age of 19 (using the childhood equations of Veale , Chandler *et al* (26) and Watson *et al* (52)), with the predicted values for non-smokers of the same height but at the age of 20 years (using the adult equations of Veale and Thompson *et al* (1)).

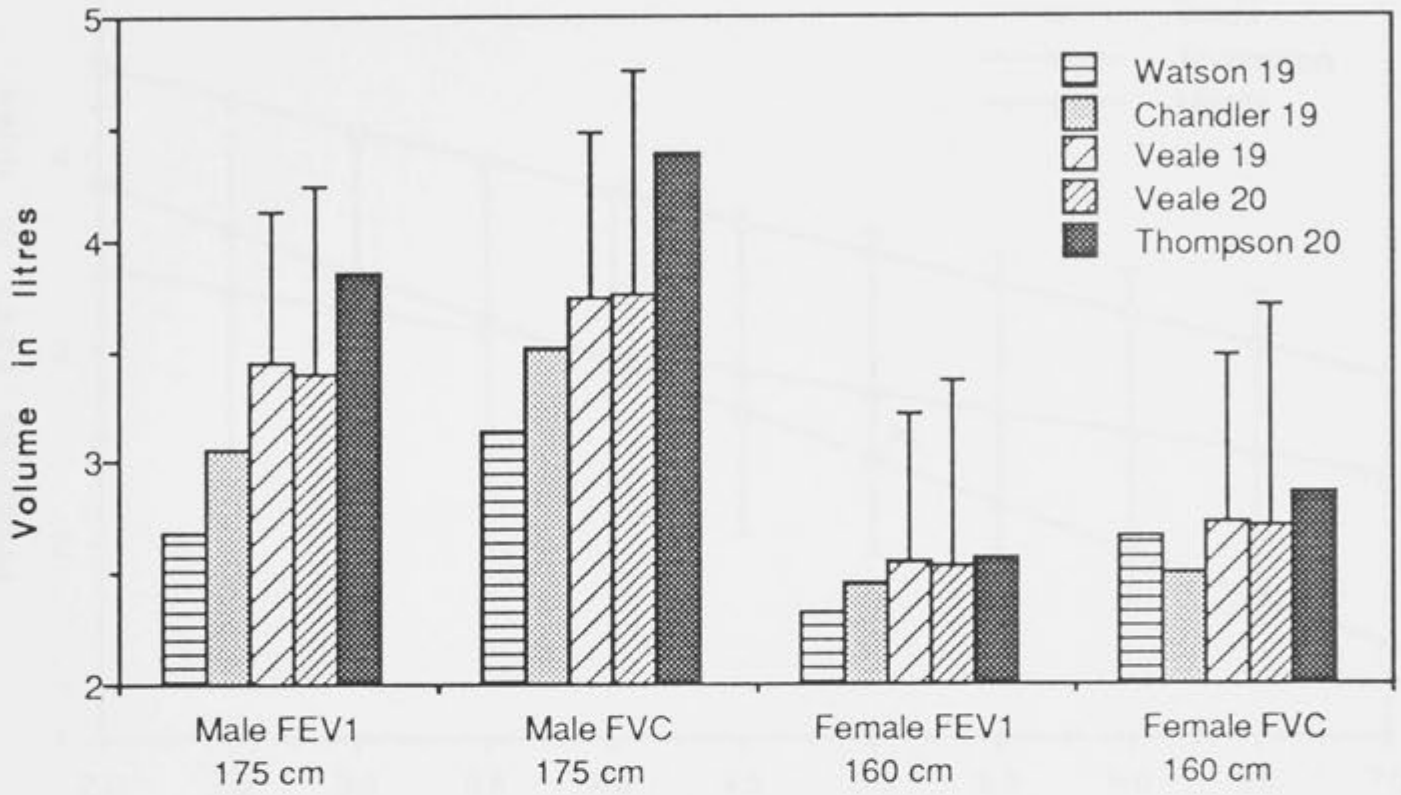
	Watson*	Chandler*	Veale	Veale	Thompson*
Age in years	19	19	19	20	20
Male 175 cm					
FEV_1	2.68	3.05	3.45	3.40	3.85
FVC	3.14	3.51	3.73	3.75	4.39
Female 160 cm					
FEV_1	2.33	2.45	2.55	2.54	2.56
FVC	2.66	2.5	2.73	2.71	2.86

* *et al*

All spirometric volumes in litres

Figure 7.2

Graph comparing the predicted spirometric values for non-smokers, at the mean height of adults, at the "transition" age of 19 (using the childhood equations of Veale , Chandler *et al* (26) and Watson *et al* (52)), with the predicted values for non-smokers of the same height at the age of 20 years (using the adult equations of Veale and Thompson *et al* (1)).



Watson 19: The predicted spirometric values at age 19 years using the equations of Watson *et al* (52).

Chandler 19: The predicted spirometric values at age 19 years using the equations of Chandler *et al* (26).

Veale 19: The predicted spirometric values at age 19 using the paediatric equations from the present study.

Veale 20: The predicted spirometric values at aged 20 using the adult equations from the present study.

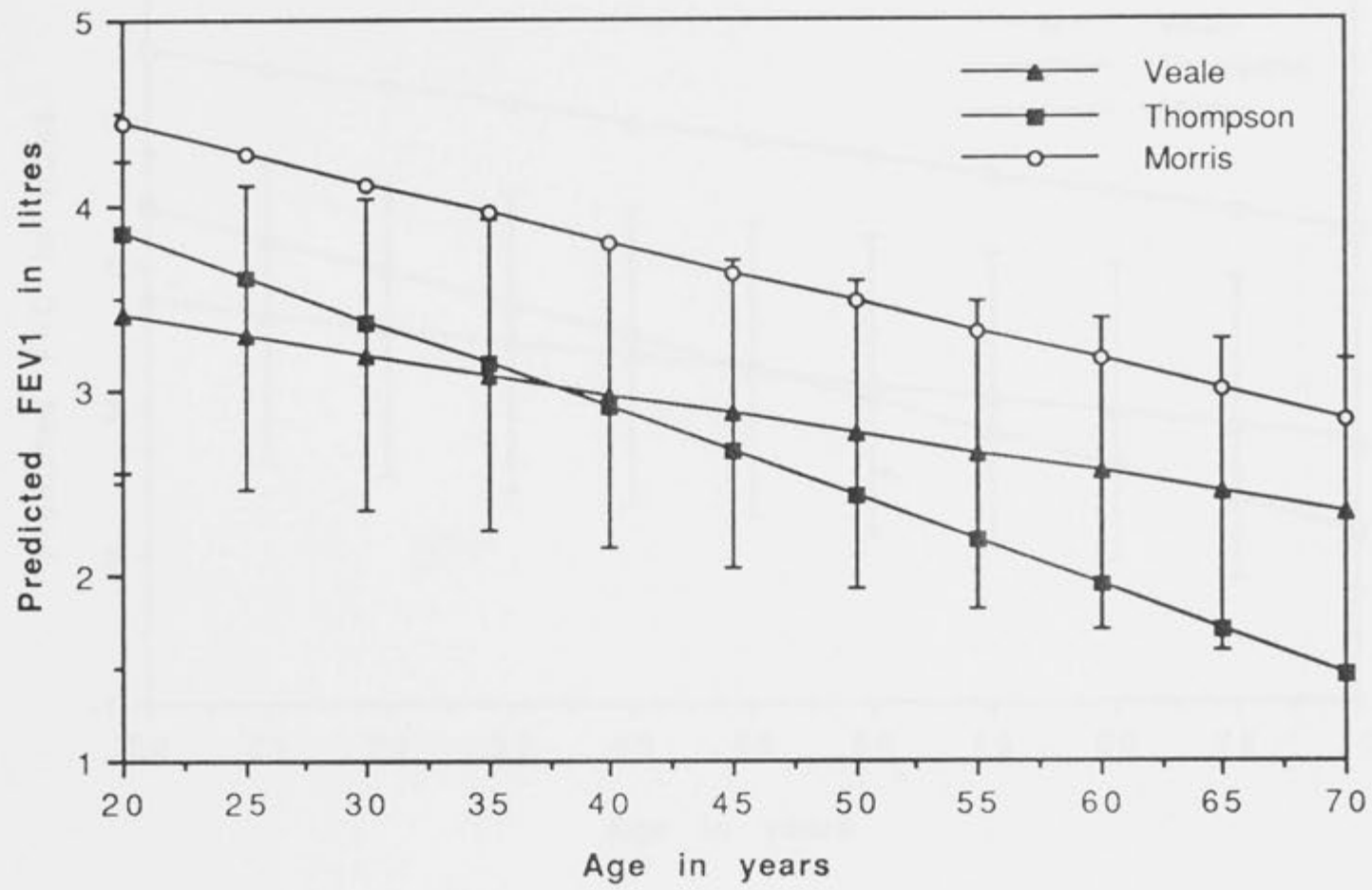
Thompson 20: The predicted spirometric values at age 20 using the equations of Thompson *et al* (1).

The T bars indicate the upper bounds of the 95% confidence interval for Veale 19 and Veale 20.

FEV_1 and FVC measured in litres.

Figure 7.3

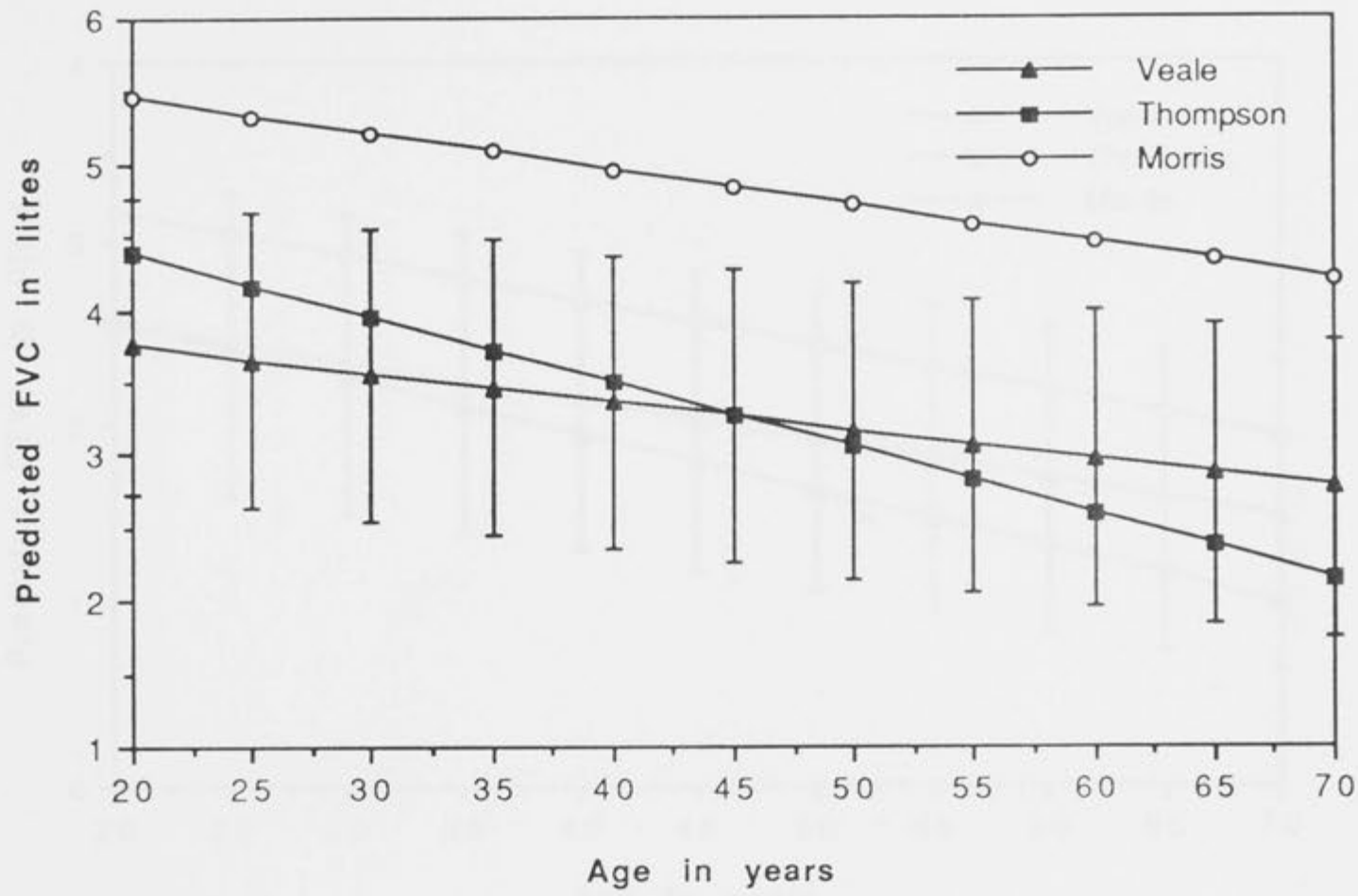
Comparison of predicted FEV_1 for 175 cm adult non-smoking males using models from the present study (Veale), Thompson *et al* (1) and Morris *et al* (93).



T bars indicate the 95% confidence intervals for Veale.

Figure 7.4

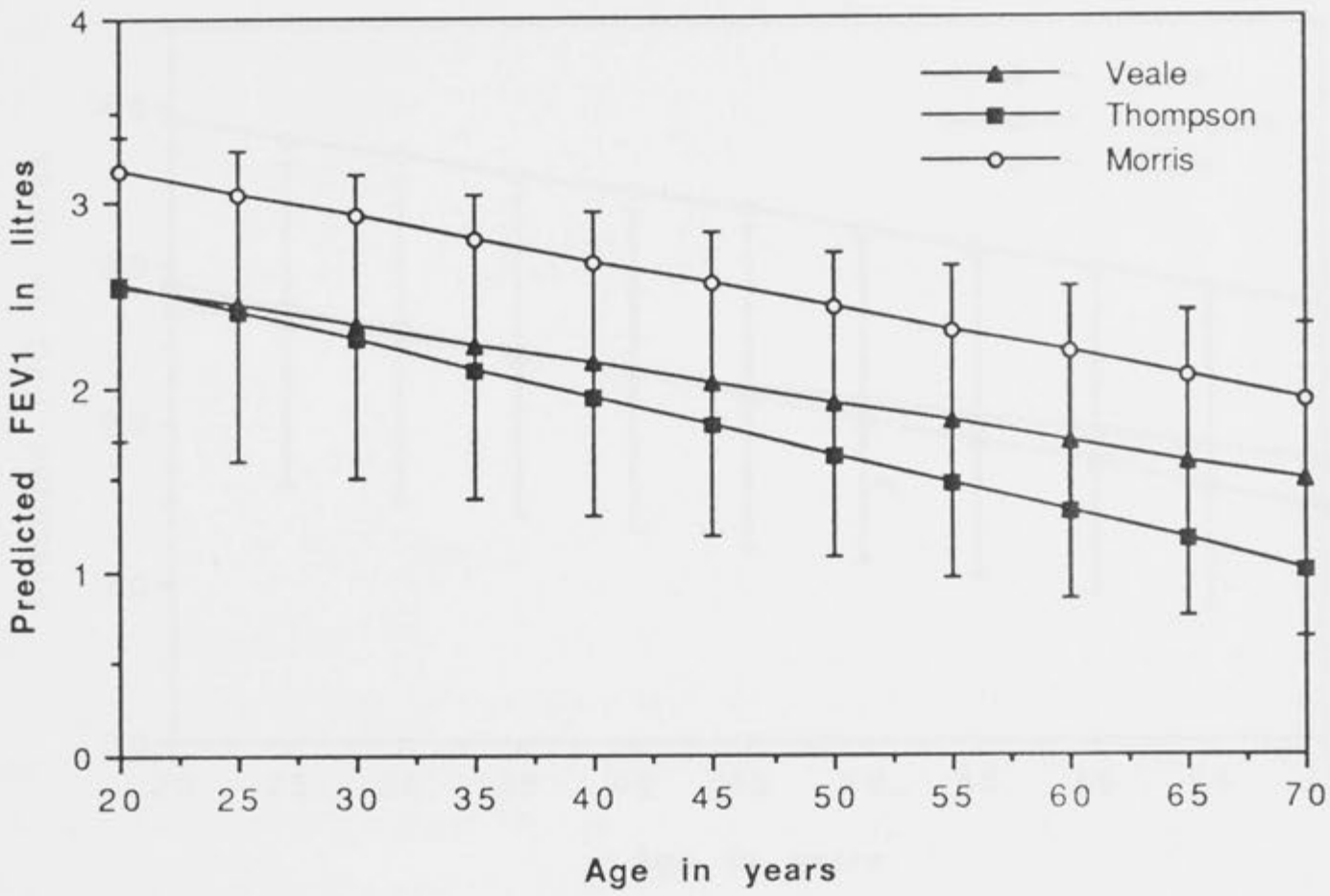
Comparison of predicted FVC for 175 cm adult non-smoking males using models from the present study (Veale), Thompson *et al* (1) and Morris *et al* (93).



T bars indicate the 95% confidence intervals for Veale.

Figure 7.5

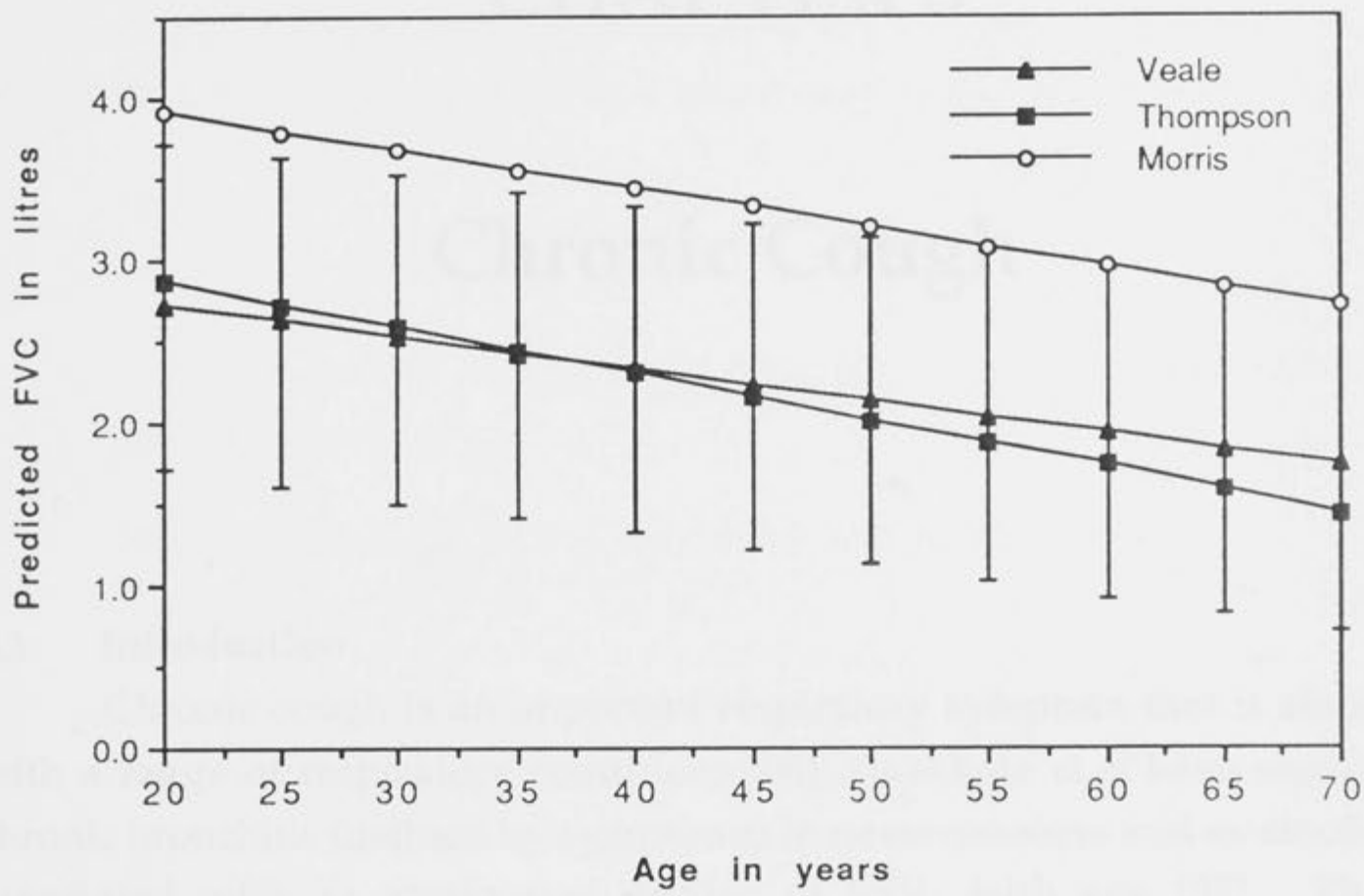
Comparison of predicted FEV_1 for 160 cm adult non-smoking females using the models from the present study (Veale), Thompson *et al* (1) and Morris *et al* (93).



T bars indicate the 95% confidence intervals for Veale.

Figure 7.6

Comparison of predicted FVC for 160 cm adult non-smoking females using models from the present study (Veale), Thompson *et al* (1) and Morris *et al* (93) .



T bars indicate the 95% confidence intervals for Veale.

CHAPTER 8

Chronic Cough

8.1 Introduction

Chronic cough is an important respiratory symptom that is associated with a range of respiratory conditions (96). Jaakkola *et al* have shown that chronic bronchitis (defined by symptoms) in never-smokers and ex-smokers, is associated with an accelerated decline of FEV_1 with age (97). Thus in epidemiological studies the prevalence of chronic cough in never- or ex-smokers may be an important indicator of the number of non-smoking people at risk of either having or developing chronic lung disease.

In Aboriginal communities the causes, prevalence and significance of chronic cough have not been adequately investigated. Such research is important because it is readily apparent when visiting Aboriginal communities that cough is a common symptom. By studying the prevalence and risk factors for chronic cough, and by examining the sensitivity and specificity of chronic cough for predicting chronic lung disease (see chapter 9), the author sought data that would allow the development of a rational approach to assessing chronic cough in isolated communities.

Many acute and chronic respiratory diseases are associated with the presence of excess bronchial secretions or pus and their presence may be detected by listening to the quality of a voluntary cough following a deep inspiration. Subjects with secretions or pus often have a loose (fruity) sounding cough. It has been shown that the detection of an audibly loose cough (positive loose cough sign), in the absence of acute respiratory tract infection, correlates with

mucus production (98). In the present study a loose cough was interpreted as indicating acute or chronic bronchial pathology.

Petrol sniffing is prevalent among adolescents and young adults in many Aboriginal communities (31). The pulmonary consequences of petrol sniffing are unknown. In CA4 petrol sniffing is common among 14-25 year olds. In this chapter the prevalence of petrol sniffing in CA4 is documented and associations with chronic cough and loose cough are examined.

8.2 Aims

The aim here was:

- To determine the prevalence of chronic cough and loose cough by age, gender, community;
- To identify the risk factors for chronic and loose cough.

8.3 Definitions

Subjects with a history of daily coughing for at least two years were defined as having chronic cough. Loose cough was considered to be present (positive loose cough test) when a the subject's cough had a moist quality.

8.4 Methods

A question was developed to determine the presence of chronic cough. Consultation with Drs Thompson and Taylor and staff from the clinics in CY1 and CY2 confirmed the author's view that the British MRC chronic bronchitis question was unsuitable for use in the present study. It was agreed that cough is a term understood by most Aboriginal people, either in English or their traditional language. Dr Taylor suggested a question be developed in the form of a story. Subjects could then be asked if they identified with the cough described in the story. The following question was the product of these discussions: "Some people cough day after day for years and years. Are you like this?" The question was readily understood when pre-tested in CY1 and CY2. The question had face validity but time did not allow reliability to be examined. The cough question was administered at interview via an interpreter who understood the purpose of the question. Subjects unable to answer the question were coded as unsure. Those coded unsure were grouped with those not having the symptom for the ensuing analysis.

Each subject was examined for the presence of a loose cough by the author who was blind to the subject's questionnaire response and current smoking status.

Two hundred and thirty six subjects were examined independently by a second respiratory physician to assess inter-observer agreement. Kappa scores (for inter-observer agreement) were calculated using the method reviewed by Sackett *et al* (75).

The method used to determine current smoking status is described in chapter 5. The method used to determine whether BHR was present is described in chapter 6. To determine if subjects sniffed petrol, a question similar to that used to determine alcohol consumption status (see figure 5.1) was used. If subjects answered "not at all" then they were regarded as non-sniffers. If they said "yes" to any of the other sniffing question options then they were identified as petrol sniffers.

The prevalence of loose cough and chronic cough was determined after stratification by age, gender, community and smoking status. An overall estimate of the prevalence of each abnormality was also determined after these data from all four communities were combined. Chi-square tests were used to assess the significance of the differences between the communities.

Following the uni-variate analyses described above, the "logistic procedure" on SAS® (92) was used to calculate adjusted odds ratios for chronic cough and loose cough. The outcome variables were coded dichotomously 0/1. The independent variables of current smoking status, petrol sniffing status, BHR and gender were also coded dichotomously 0/1. Age was treated as a continuous variable measured in years. Dummy dichotomous variables were created to identify CY2, CA3 and CA4. The parameters were calculated by iteratively reweighted least squares and the model fit was assessed using maximum likelihood. Coefficients were regarded as significant if $p < 0.05$. Age, gender, BHR, current smoking status, current petrol sniffing status and the dummy variables for CY2, CA3 and CA4 were all included in the initial models. Current asthma was not considered separately as a risk factor for chronic cough or loose cough because it correlated highly with BHR (see chapter 6). The least statistically significant variables were removed from the models one at a time.

8.5 Results

Tables 8.1 to 8.3 show the prevalence of chronic and loose cough by age, gender, community and smoking status. Figures 8.1 and 8.2 show these data by age, gender and smoking status.

8.5.1 Chronic Cough

In males the prevalence of chronic cough increased with age and was similar in smokers and non-smokers (see figure 8.1). There was no significant regional variation in the prevalence of chronic cough in male smokers or non-smokers.

In females the prevalence of chronic cough increased with age and was higher in smokers than in non-smokers (see figure 8.2). There was significant regional variation in the prevalence of chronic cough in 20-84 year old female smokers (chi-square $p < 0.05$) (see table 8.3).

Despite pre-testing, 14% of 5-13 year old children (or their parent or guardian) and 2% of 14-84 year olds were confused ("unsure") about the chronic cough question. The prevalence of loose cough in "unsure" subjects, was not significantly different from those without chronic cough.

8.5.2 Loose Cough

In males the prevalence of loose cough had an early peak in 14-19 year olds and then, following a "trough" in 20-39 year olds, increased in prevalence with age (see figure 8.1). The prevalence of loose cough was higher in smokers than non-smokers. In male non-smokers there was significant regional variation in the prevalence of loose cough at all ages (chi-square $p < 0.05$) (see tables 8.1 to 8.3). Among male smokers there was no significant regional variation in the prevalence of loose cough.

In females the prevalence of loose cough with age followed a similar pattern to that of the males although the difference between smokers and non-smokers was greater in females (see figure 8.2). In female non-smokers there was significant regional variation in the prevalence of loose cough among 14-19 year olds and 20-84 year olds (chi-square $p < 0.05$) (see tables 8.2 and 8.3). In female smokers there was no significant regional variation in the prevalence of loose cough.

8.5.3 Inter-observer Agreement for Loose Cough

Two hundred and thirty subjects were examined independently for the presence of a loose cough. The Kappa statistic for the level of agreement was 0.47 (see table 8.4).

8.5.4 Petrol Sniffing

The prevalence of self-reported petrol sniffing in 15-25 year olds subjects in CA4 was 44% in males and 33% in females (see table 8.5). In CA4 there were

no self-reported sniffers under the age of 14 years or over the age of 25 and petrol sniffing was unknown in the other communities at the time of the present study. Table 8.6 shows the self-reported sniffing pattern among male and female sniffers.

8.5.5 Adjusted Odds Ratios for Chronic and Loose Cough

The adjusted odds ratios for chronic cough and loose cough are shown in table 8.7. Age, petrol sniffing and cigarette smoking were all significantly associated with an increase in the odds for chronic cough. There was no significant regional variation in the prevalence of chronic cough. Age , petrol sniffing and cigarette smoking were also significantly associated with an increase in the odds for loose cough. There was significant regional variation in the prevalence of loose cough because being from CA3 was associated with a reduction in the odds for having this abnormality.

Table 8.1

Prevalence of chronic cough and loose cough in 5-13 year olds by community and gender.

	CY1	CY2	CA3	CA4	All	95% CI
Males						
Non-smokers (N)	47	14	42	22	125	N/A
Smokers (N)	-	-	-	-	-	-
Chronic Cough						
Non-smokers	8.5	0.0	0.0	9.1	4.8¤	1.0-8.6
Smokers	-	-	-	-	-	-
Loose Cough						
Non-smokers	10.6	14.3	16.7	45.5	19.2**	12.3-26.1
Smokers	-	-	-	-	-	-
Females						
Non-smokers (N)	44	10	37	25	116	N/A
Smokers (N)	-	-	-	-	-	-
Chronic Cough						
Non-smokers	9.8	11.1	2.7	4.0	6.3¤	1.8-10.8
Smokers	-	-	-	-	-	-
Loose Cough						
Non-smokers	13.6	20.0	13.5	24.0	16.4¤	9.6-23.1
Smokers	-	-	-	-	-	-

Prevalence expressed as the percentage found in smokers and non-smokers. The denominators N (for non-smokers and smokers) are located in the first row of each gender stratum. As there were only four smokers in this age group their findings are included with the non-smokers.

¤ Chi-square for the difference between communities not significant.

** Chi-square for the difference between communities $p < 0.01$.

Table 8.2

Prevalence of chronic cough and loose cough in 14-19 year olds by community, gender and smoking status.

	CY1	CY2	CA3	CA4	All	95% CI
Males						
Non-smokers (N)	10	4	27	12	53	N/A
Smokers (N)	10	2	18	13	43	N/A
Chronic Cough						
Non-smokers	0.0	0.0	7.4	25.0	9.4¤	1.5-17.2
Smokers	10.0	0.0	16.7	0.0	9.3¤	0.6-17.9
Loose Cough						
Non-smokers	20.0	0.0	11.1	50.0	20.8*	9.8-31.7
Smokers	20.0	50.0	27.8	38.5	30.2¤	16.4-43.9
Females						
Non-smokers (N)	11	1	27	14	53	N/A
Smokers (N)	13	3	-	7	23	N/A
Chronic Cough						
Non-smokers	0.0	0.0	4.0	7.1	3.9¤	0.0-9.2
Smokers	30.8	0.0	-	14.3	22.7¤	5.0-40.0
Loose Cough						
Non-smokers	0.0	0.0	3.7	28.6	9.4*	1.5-17.2
Smokers	23.1	33.3	-	42.9	30.4¤	11.6-49.2

Prevalence data expressed as the percentage found in smokers and non-smokers. The denominators N (for non-smokers and smokers) are located in the first row of each gender stratum.

¤ Chi-square for the difference between communities not significant.

* Chi-square for the difference between communities $p < 0.05$.

Table 8.3

Prevalence of chronic cough and loose cough in 20-84 year olds by community, gender and smoking status.

	CY1	CY2	CA3	CA4	All	95% CI
Males						
Non-smokers (N)	35	8	31	35	109	N/A
Smokers (N)	73	21	47	29	170	N/A
Chronic Cough						
Non-smokers	14.7	12.5	6.5	17.1	13.0¤	6.6-19.3
Smokers	15.1	10.0	10.6	13.8	13.0¤	7.9-18.0
Loose Cough						
Non-smokers	2.9	25.0	6.5	37.1	16.5***	9.5-23.4
Smokers	26.0	19.0	21.3	41.4	26.5¤	19.8-33.1
Females						
Non-smokers (N)	57	9	102	59	227	N/A
Smokers (N)	78	28	1	18	125	N/A
Chronic Cough						
Non-smokers	7.0	0.0	4.9	13.6	7.5¤	4.0-10.9
Smokers	14.1	11.1	100.0	5.6	12.9*	7.0-18.8
Loose Cough						
Non-smokers	10.5	0.0	7.8	25.4	12.8**	8.4-17.1
Smokers	33.3	32.0	100.0	22.2	32.0¤	23.8-40.0

Prevalence data expressed as the percentage found in smokers and non-smokers. The denominators N (for non-smokers and smokers) are located in the first row of each gender stratum.

¤ Chi-square for the difference between communities not significant.

* Chi-square for the difference between communities $p < 0.05$.

** Chi-square for the difference between communities $p < 0.01$.

*** Chi-square for the difference between communities $p < 0.001$.

Table 8.4

Inter-observer agreement for loose cough (230 subjects).

Chance*	Observed**	Kappa Statistic	95% CI***
67.5%	83%	0.47 (SE 0.072)	0.3 - 0.6

* = percent agreement expected by chance

** = percent agreement observed

*** = 95% Confidence Interval of Kappa Statistic

Table 8.5

Petrol sniffing in 14-25 year olds in CA4.

	N	Number sniffers	% sniffers
Males	39	17	44
Females	42	14	33

Table 8.6

Self-reported sniffing pattern among 14-25 year old CA4 petrol sniffers as a percentage of sniffers by gender.

	Males	Females
A little bit	18.0	57.0
A moderate amount	12.0	21.5
Heavily at times	41.0	0.0
Every day	29.0	21.5

Table 8.7

Adjusted odds ratios for chronic cough and loose cough in 5-84 year olds.

	Odds ratio	95% CI	p value
Chronic Cough			
Age	1.02	1.01-1.03	< 0.01
Petrol Sniffers	3.5	1.43-8.68	< 0.01
Cigarette Smokers	1.68	1.09-2.58	< 0.05
Loose Cough			
Age	1.01	1.00-1.02	< 0.05
Petrol Sniffers	3.67	1.73-7.79	< 0.001
Cigarette Smokers	1.99	1.44-2.76	< 0.001
CA3	0.60	0.41-0.89	< 0.05

Odds ratio (OR) for age = the increase in odds for outcome with each incremental year of age.

OR for petrol sniffers = the increase in odds for outcome if the subject was a petrol sniffer.

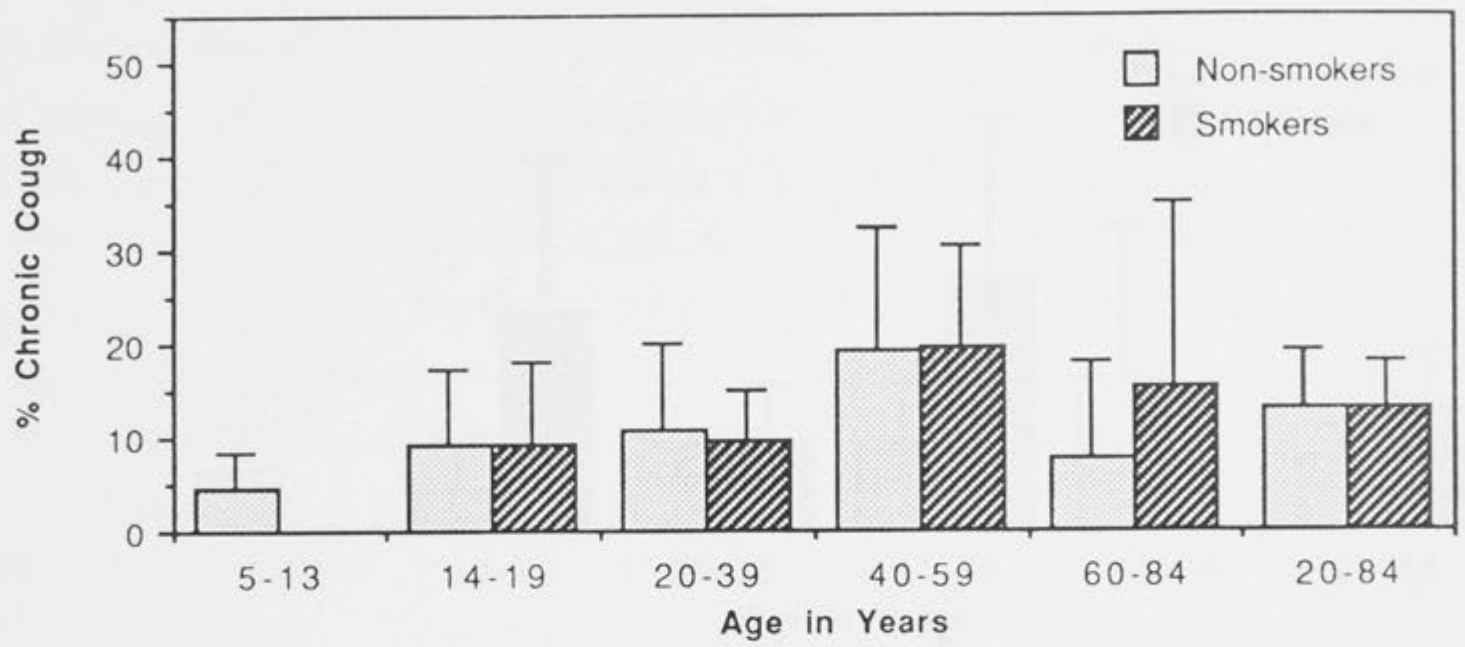
OR for smokers = increase in odds for outcome if the subject was a smoker.

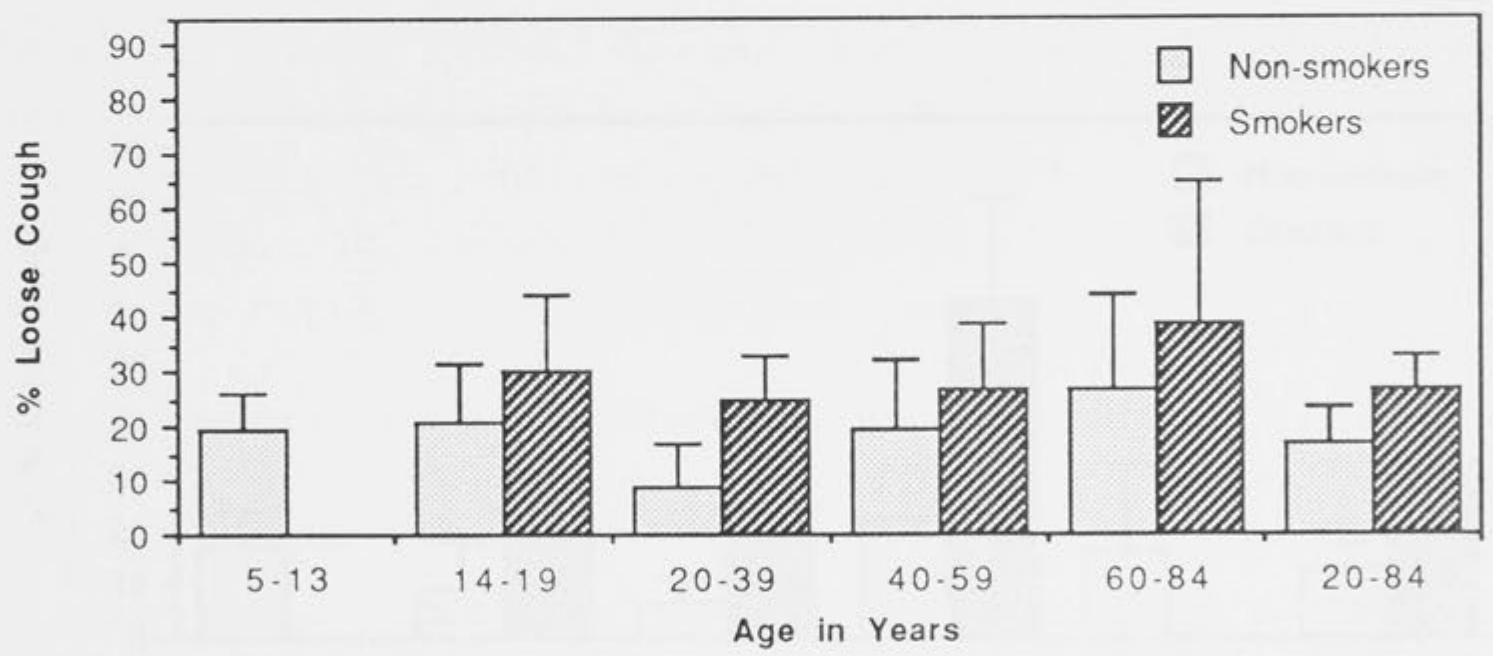
OR for CA3 = the reduction in odds for outcome for subjects tested in CA3.

The odds ratios for each variable in the model were adjusted for confounding by each other.

Figure 8.1

Chronic cough and loose cough in non-smoking and smoking males by age.

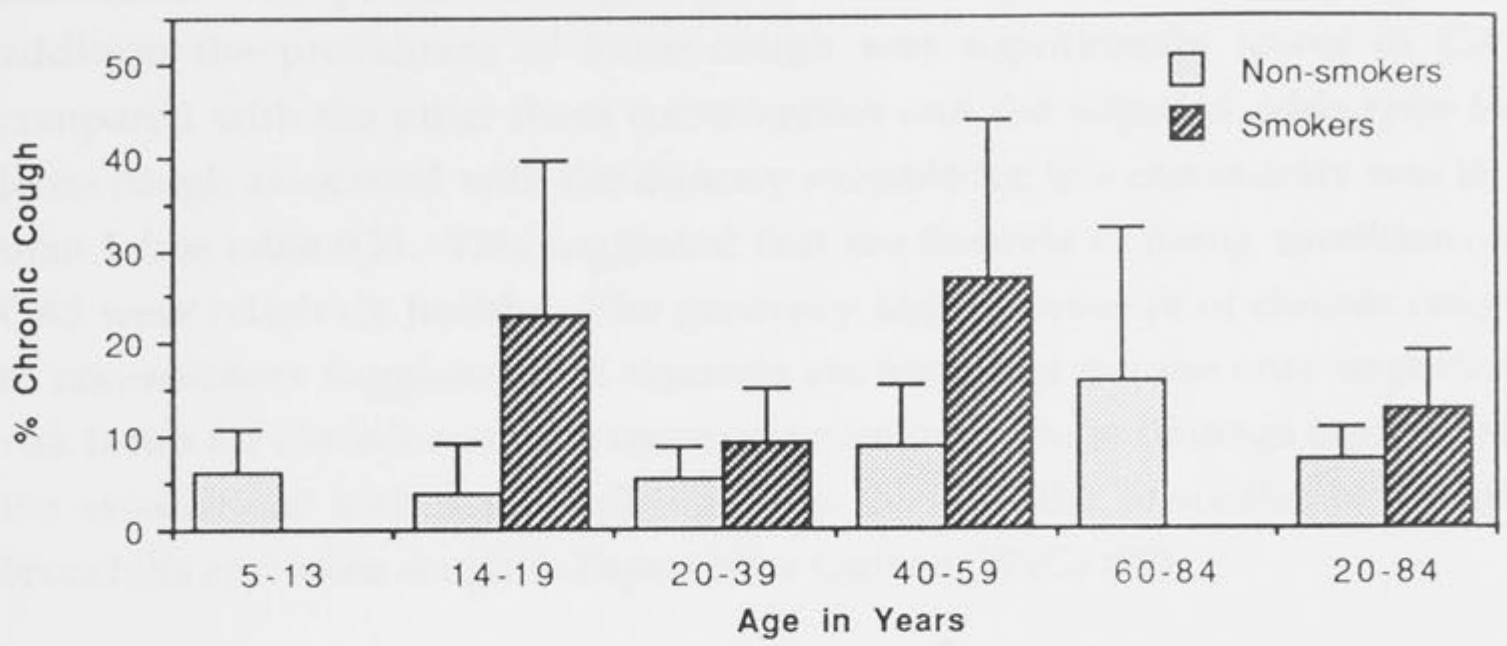


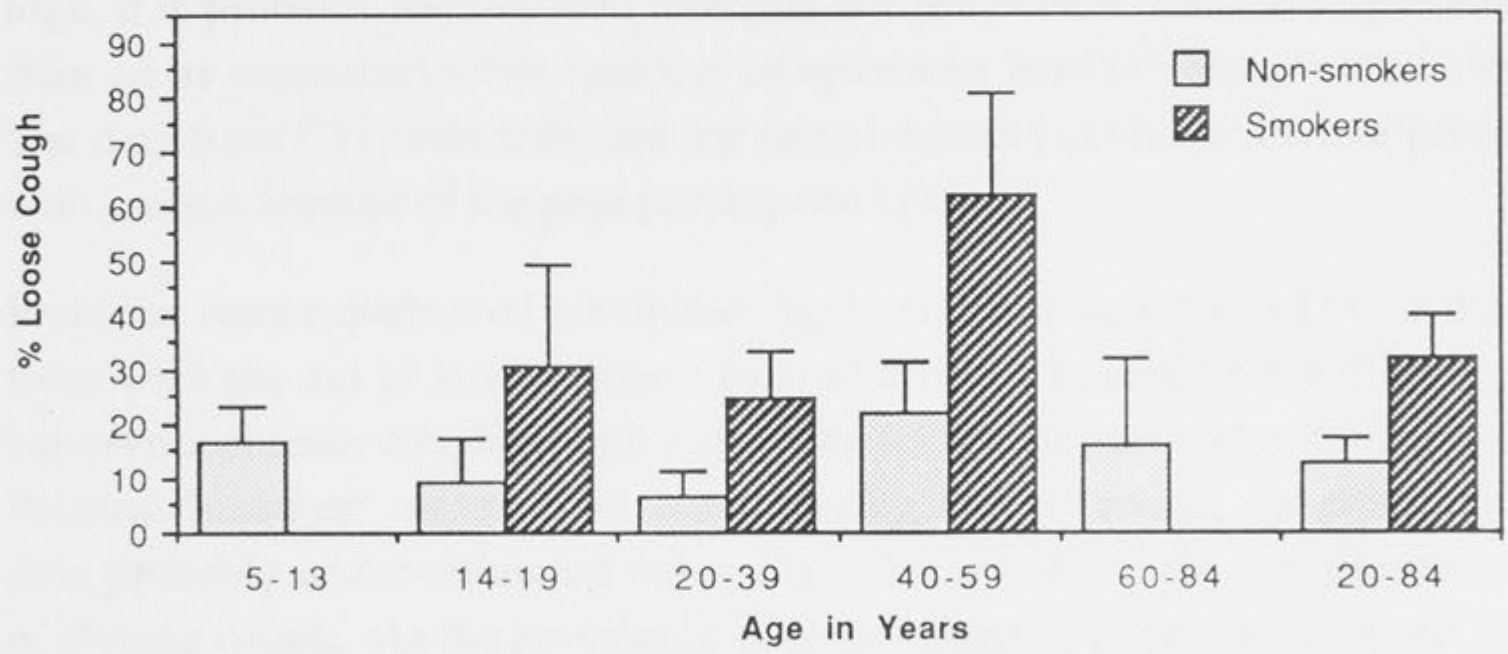


T bars indicate the upper limits of the 95% confidence interval of the estimates.

Figure 8.2

Chronic cough and loose cough in non-smoking and smoking females by age.





T bars indicate the upper limits of the 95% confidence interval of the estimates.

8.6 Discussion

The pertinent findings were that chronic and loose cough were associated with petrol sniffing, cigarette smoking and increasing age. In addition the prevalence of loose cough was significantly lower in CA3 compared with the other three communities and the adjusted odds ratio for loose cough associated with the dummy variable for this community was less than 1 (see table 8.7). This suggested that the lifestyle or living conditions in CA3 were relatively healthy. The generally high prevalence of chronic cough in non-smokers suggested that cigarette smoking was not the only important risk factor for chronic cough in these communities. These findings (apart from the associations with petrol sniffing) were very similar to studies of chronic bronchitis and loose cough in Papua New Guinea (PNG) (79).

These data in tables 8.1 to 8.3 highlight the problem of low numbers and hence limited statistical power for researchers of small communities. Despite studying over one thousand people, stratification by age, gender, community and smoking status, resulted in many cells with low numbers. This was particularly the case in CY2. It is recognised that in this situation tests such as the chi-square test may be inaccurate, and that logistic regression models may be unstable (99, 100). In CY1, CA3 and CA4, where the participation rates were high, it is probably legitimate to interpret the prevalence data literally (rather than an as estimates) when making comparisons between the communities. The data from CY2 were included for completeness but should be interpreted with caution because of the poor participation rate.

Problems were experienced administering the cough question to 5-13 year olds. Even with the aid of interpreters it proved difficult to explain the difference between a chronic cough (cough every day for years) and a recurrent cough. Because "unsures" were coded as not having chronic cough, the prevalence data probably under-estimated rather than over-estimated the true prevalence of chronic cough. As the prevalence of loose cough in those unsure about the chronic cough question was similar to those who denied chronic cough, the decision to group unsures with negatives has probably not introduced important bias.

A person meeting the criteria for chronic cough in the present study was likely also to meet the British MRC criteria for chronic bronchitis because someone with daily coughing for two years is likely to have serious lung disease or chronic bronchial irritation. By inference the prevalence of chronic bronchitis

(MRC definition) in the present study was probably higher than the prevalence of chronic cough. This assertion allows some comparison with epidemiological studies of chronic bronchitis.

Petrol sniffing (see tables 8.5 and 8.6) was more prevalent and heavier among males than females. Although the scale for petrol sniffing behaviour (like the one used for alcohol consumption) is not validated nor standardised and combines an assessment of volume and frequency, the findings have some face validity and attest to the prevalence of the petrol sniffing in CA4.

8.6.1 Chronic Cough

The overall prevalence of chronic cough in the 5-13 year old children (<7%) was difficult to interpret because of the low numbers and doubts about the validity of the question in this age group. In Southhampton (UK) Clifford *et al* (101) found that 18.5% of seven year old non-asthmatic children (n=1,242), and 8.7% of eleven year olds (n=1,218), reported recurrent coughing unrelated to upper respiratory tract infections. The lack of respiratory symptom studies in other Aboriginal children, make it impossible to make any comparisons regarding the prevalence of chronic cough in this age group.

The prevalence of chronic cough in non-smoking 14-19 year old males in CA4 (see table 8.2) was high when contrasted with the other three communities. Non-smoking adolescents without asthma were expected to have a low prevalence of chronic cough because they were beyond the age when childhood bronchial infections are prevalent and they didn't smoke. This finding suggested that chronic lung disease or bronchial irritants other than smoking were prevalent in CA4.

The prevalence of chronic cough in adult non-smokers was high (see table 8.3) when contrasted with the findings in Busselton WA in 1966 where the prevalence of chronic bronchitis was 2-3% in non-smokers (102). In addition, the prevalence of chronic cough in adult non-smokers was relatively low in CA3, when contrasted with the other three communities. This finding was interesting because in chapter 9 it is demonstrated that the prevalence of chronic lung disease was also relatively low in this community.

The multivariate analysis revealed that cigarette smoking, petrol sniffing and age were independent risk factors for chronic cough (see table 8.7). Petrol sniffing may partially explain the high prevalence of chronic cough in CA4 14-19 year old non-smokers (see table 8.2). However, the high prevalence of

chronic cough in non-smokers in the communities where petrol sniffing did not occur (see table 8.3), suggested that risk factors for chronic cough other than petrol sniffing and cigarette smoking were present.

8.6.2 Loose Cough

The inter-observer agreement (Kappa statistic 0.47) achieved for loose cough was comparable with the findings of Withey *et al* (103). Withey *et al* examined the repeatability of the loose cough sign in 60 subjects in whom the prevalence of loose cough ranged from 17% to 20%, and achieved a kappa score of 0.53 (SE of 0.24). Kappa scores ranging from 0.41 to 0.60 were considered by Ladis and Koch (76) to indicate moderate strength of agreement. The findings in the present study confirm that the loose cough sign is an observation which has a moderate level of inter-observer agreement and is suitable for use in epidemiological studies of Aboriginal communities.

The prevalence of loose cough (see tables 8.1 to 8.3) was similar to the findings of Torzillo *et al* (23) and Cullen (102). Torzillo *et al* examined 1,287 Aboriginal people in the Pilbara (WA) and found a loose cough in 15% of 0-19 year olds and 20% of adults. It is not known what proportion of the Pilbara subjects were smokers. In 1968 Cullen found that 12% of 538 predominantly Caucasian males aged 20-39 (and 31% of 1100 males over the age of 40) from Busselton, WA had a positive loose cough sign. The prevalence of smoking in the Busselton males was 51% but the prevalence of loose cough by smoking status was not reported. The prevalence of loose cough in the present study was also comparable to the results of studies in PNG (79).

The multivariate analysis showed that cigarette smoking, petrol sniffing and age were independent risk factors for loose cough (see table 8.7). However the generally high prevalence of loose cough in non-smokers and non-sniffers (see table 8.3) suggested that risk factors for loose cough, other than smoking and sniffing, were important in these communities.

Among adults, the regional variation in the prevalence of loose cough reflected the regional variation in chronic cough (see table 8.3). The multivariate analysis showed that subjects in CA3 were 40% less likely to have loose cough (controlling for age, cigarette smoking and petrol sniffing) than subjects in the other three communities (see table 8.7). This finding was non-specific because loose cough can be secondary to bronchial irritation from inhaled particulate matter, bronchial infection, pulmonary infection or chronic lung disease.

Nevertheless it does suggest that the people of CA3 were relatively advantaged compared with those in the other three communities in terms of lung health .

8.7 Conclusion

- Age, petrol sniffing and cigarette smoking were risk factors for chronic cough and loose cough.
- The prevalence of chronic cough and loose cough was high among people who did not sniff petrol or smoke cigarettes suggesting that other lifestyle or environmental risk factors for chronic lung disease were present.
- The adjusted odds ratio for loose cough in people from CA3 suggested that the lifestyle or environment in that community was relatively healthy.
- Petrol sniffing among adolescents in CA4 was prevalent.
- The loose cough sign can be used with a moderate level of inter-observer agreement in epidemiological studies of Aboriginal communities.

CHAPTER 9

Chronic Lung Disease

9.1 Introduction

Studies of chronic lung disease (CLD) in Aborigines are needed because the relative risk of death from chronic obstructive pulmonary disease (COPD) and pneumonia is high in comparison with non-Aboriginal Australians (27). Also, many health professionals who have worked in Aboriginal communities believe bronchiectasis and COPD are prevalent.

9.1.1 Definitions

CLD was the term used in the present study to refer to all lung diseases associated with abnormal lung function (ALF). COPD is the diagnosis generally given to people who have chronic irreversible airflow obstruction. Cigarette smoking is a well recognised cause of COPD. The airflow obstruction developed by cigarette smokers is probably caused by inflammation and fibrosis in respiratory bronchioles, emphysema or both (45). Pathological processes that lead to fibrosis of the pulmonary parenchyma or pleura, or decreased compliance of the chest wall can lead to a reduced total lung capacity and non-obstructive ventilatory defects (so called restrictive ventilatory defect) (48). In the present study subjects with non-obstructive ventilatory defects were diagnosed as having non-obstructive lung disease (NOLD).

A variety of definitions have been used to define COPD in epidemiological studies of non-Aboriginal people (45). In accordance with ATS guidelines for interpreting spirometric function, subjects with a FEV_1, FVC or FEV_1/FVC ratio greater than two standard deviations below normal were regarded as having

abnormal lung function (ALF) (48). Subjects with ALF and an abnormal FEV_1/FVC ratio were diagnosed as having COPD. Subjects with ALF and a normal FEV_1/FVC ratio were diagnosed as having NOLD. In this chapter, data regarding the prevalence of COPD and NOLD are presented and hypotheses regarding the potential causes are explored.

9.1.2 Potential Risk Factors for CLD

Studies in other populations suggest that lower respiratory tract infections in childhood, cigarette smoking, alcohol consumption and adverse living conditions (an unhealthy living environment) are all risk factors for the development of CLD. The findings regarding petrol sniffing from the present study (see chapter 8) suggest that petrol vapour inhalation may also be a risk factor for CLD. These possibilities are discussed further below.

Gold *et al* compared spirometric function among Caucasian male non-asthmatic children with and without a history of pneumonia or hospitalisation for respiratory illness prior to the age of two years (5). Those children with the positive history of respiratory illness had mean reductions of FEV_{25-75} by 20% and FEV_1 by 6% up to thirteen years later. Whether or not childhood respiratory illness is a risk factor for COPD in adults remains controversial (104). It is not known if pneumonia in Aboriginal children is associated with spirometric abnormalities or if it is a risk factor for COPD in adults. However clinical and radiological studies of Aboriginal adults and children have implicated pneumonia as a risk factor for the development of bronchiectasis so severe that it caused total lung destruction (see chapter 2). Therefore it is likely that a proportion of children with bronchiectasis have ALF that persists into adulthood.

The prevalence of cigarette smoking in many Aboriginal communities is high when compared with that found among non-Aboriginal Australians (see chapter 5). As smoking is an accepted cause of COPD in other populations (45) it is probable that it is also causes COPD in Aborigines. In Aboriginal communities cigarette smoking could be influencing the prevalence of CLD by augmenting the rate of decline of FEV_1 with age in adults (45) or by influencing the growth of FEV_1 in adolescents (105, 106).

Alcohol consumption has been associated with COPD in cohort studies of lung function although the mechanism underlying this association is unclear (107). Although alcohol use is prevalent in many Aboriginal communities, whether this factor has a role in the aetiology of CLD in Aborigines is unknown.

The outdoor living environment for many Aboriginal people is polluted by high levels of smoke from wood fires and particulate matter in the form of dust (11, 28). Whilst indoor smoke from wood fires has been associated with lower respiratory tract infections in American Indian children (108) there is little evidence that domestic smoke exposure is directly associated with CLD (109). Little is known about the effects of chronic inhalation of non-specific dust (110) but it has been shown that some dusts are associated with COPD (111). Although domestic smoke exposure and environmental dust exposure are possible risk factors for CLD in Aboriginal communities, it was beyond the scope of the present study to measure exposure to these agents and therefore to assess their impact specifically.

The quality of the indoor living environment and housing may also influence the development of CLD but there is little evidence to support this at present (112). An assessment of any regional variation in the prevalence of CLD in this study provides an opportunity to generate hypotheses regarding the importance of environmental factors in the genesis of CLD. However assessing the contribution of specific environmental factors is complicated by the difficulty of identifying the important elements, quantifying exposure and controlling for confounders (110).

Petrol sniffing is a prevalent activity among adolescents in many communities and some sniffers spend up to 24 hours a day inhaling fresh petrol vapour (31). In chapter 8 it was shown that petrol sniffing was a risk factor for chronic and loose cough. Petrol sniffing may also be a risk factor for CLD. In this chapter the relationship between petrol sniffing and ALF is examined.

9.1.3 Respiratory Symptoms, Signs and CLD

Although respiratory symptoms and signs are common in Aborigines (see chapter 8) their sensitivity and specificity for diagnosing CLD is not known. It would be useful for health service staff in remote communities if clinical findings could be used to predict ALF as this would assist in the identification of people with CLD. For this reason the clinical associations of CLD were examined.

9.2 Aims

The aim here was:

- To determine the prevalence of COPD and NOLD by age, gender and community;
- To identify risk factors for COPD and NOLD;
- To determine the clinical features of COPD and NOLD.

9.3 Methods

The collection of respiratory symptom and sign data is described in chapters 6 and 8. The performance of spirometric tests and the method used to develop equations to predict FEV_1 and FVC are described in chapters 6 and 7. Subjects who were unable to blow reproducible forced expirations (ATS criteria) were regarded as having technically unsatisfactory spirometric tests (91).

To assess bias resulting from inability to perform spirometric tests to ATS criteria (113), the proportion of subjects with technically unsatisfactory spirometric tests was documented by age and community. The following factors in subjects with technically satisfactory and unsatisfactory spirometric tests were examined in four age strata: gender, current smokers, ex-smokers, chronic cough, recent wheeze, loose cough, rhonchi and crepitations.

To explore the risk factors for ALF the prevalence of people who were current, ex- or ever-smokers, petrol sniffers and current alcohol users were determined for those with and without COPD, NOLD and CLD. Also evidence for regional variation in the prevalence of ALF was sought.

To explore the clinical features of COPD and NOLD the prevalence of chronic cough, recent wheeze, loose cough, rhonchi, crepitations, BHR and asthma were examined for those with and without ALF. Chi-square or Mantel Haenszel chi-square tests were performed to assess the significance of any differences between the groups.

Following the univariate analyses described above, logistic regression using the "logistic procedure" on SAS® (92) was used to calculate adjusted odds ratios for NOLD, COPD and CLD. The outcome variable was coded dichotomously. Current smoking, ex-smoking, ever-smoking, sniffing of petrol, BHR and gender were coded dichotomously and used as independent variables. Age in years was treated as a continuous variable. Dummy dichotomous variables were created to identify CY1, CA3 and CA4. Variables were created to

determine if chronic cough, recent wheeze, loose cough, crepitations and rhonchi were associated with CLD. The coefficients for each variable were calculated by iteratively reweighted least squares and the model fit was assessed using maximum likelihood. Coefficients were regarded as significant where $p < 0.05$. Age, gender, BHR, the dummy variables for the communities, and the three smoking related variables (one at a time to avoid multiple collinearity) were all included in the initial models. The least statistically significant variables were removed from the models sequentially. Current asthma was not considered separately as a risk factor for CLD because it is highly correlated with BHR (see chapter 6). Tests for interactions were not performed to keep the models parsimonious and because clinical interpretation of significant coefficients would have been problematic.

9.4 Results

9.4.1 FEV_1/FVC Ratio in Adults and Children

Table 9.1 presents the mean FEV_1/FVC ratios in the reference population (see chapter 7) stratified by age. The mean FEV_1/FVC ratio in 7-19 year old children was 93.7% (SD 5.1%). The mean FEV_1/FVC ratio in 20-70 year olds was 89.8 % (SD 7.1%). Using the definition outlined above it follows that 7-19 year olds with FEV_1/FVC ratios below 83.5% and 20-70 year olds with FEV_1/FVC ratios below 75.6% had ALF.

9.4.2 Prevalence of Unsatisfactory Spirometric Function

The prevalence of subjects with technically unsatisfactory spirometric function stratified by age and community is shown in table 9.2. In sixty subjects either the FEV_1 or FVC was not reproducible but the other index was satisfactory. These people were included in the technically satisfactory group.

Table 9.3 shows the prevalence of current smokers, ex-smokers, chronic cough, recent wheeze, loose cough, rhonchi and crepitations in subjects with technically satisfactory and unsatisfactory spirometric tests stratified by age.

Table 9.1

Mean observed FEV_1/FVC ratios for children and adults in the reference population.

		FEV_1/FVC ratio	
Age (Years)	N	Mean	SD
Children			
7-9	56	94.4	4.4
10-12	73	92.8	5.7
13-15	63	93.8	5.1
16-19	69	93.8	4.9
7-19	**261**	**93.7**	**5.1**
Adults			
20-30	146	90.9	5.9
31-40	50	90.2	6.6
41-50	82	87.9	7.6
51-60	39	88.3	9.3
61-70	14	87.1	9.1
20-70	**331**	**89.8**	**7.1**

Table 9.2

Prevalence of technically unsatisfactory spirometric tests by age and community.

Age group	N*	TU**	% TU†	TS#
7-19				
CY1	164	30	18	134
CY2	48	14	29	34
CA3	153	7	5	146
CA4	86	4	5	82
All	451	55	12	396
20-39				
CY1	161	13	8	148
CY2	59	14	24	45
CA3	111	2	2	109
CA4	81	2	2	79
All	412	31	7	381
40-59				
CY1	85	13	15	72
CY2	30	11	37	19
CA3	54	1	2	53
CA4	50	3	6	47
All	219	28	13	191
60-84				
CY1	32	9	28	23
CY2	9	7	88	2
CA3	20	1	5	19
CA4	18	3	17	15
All	79	20	25	59

* Number of participants by community.

** Number of participants with technically unsatisfactory spirometric tests.

† % of subjects with technically unsatisfactory spirometric tests.

Number of participants with technically satisfactory tests.

Table 9.3
Prevalence of exploratory variables (as a percentage) in subjects with and without technically satisfactory spirometric tests by age.

	Technically satisfactory	Technically unsatisfactory	Chi-square*
7-19 Year olds	N=396	N=55	
Females	47	47	-
Current smokers	17	4	$p < 0.05$
Ex-smokers	1	1	-
Chronic cough	7	7	-
Recent Wheeze	4	0	-
Loose Cough	18	24	-
Rhonchi	5	4	-
Crepitations	2	2	-
20-39 Year olds	N=381	N=31	
Females	60	64	-
Current smokers	53	61	-
Ex-smokers	8	13	-
Chronic cough	8	17	-
Recent Wheeze	8	13	-
Loose Cough	16	10	-
Rhonchi	3	0	-
Crepitations	4	3	-
40-59 Year olds	N=191	N=28	
Females	54	46	-
Current smokers	41	39	-
Ex-smokers	16	32	$p < 0.05$
Chronic cough	16	32	$p < 0.05$
Recent Wheeze	14	18	-
Loose Cough	28	32	-
Rhonchi	9	14	-
Crepitations	7	18	$p < 0.05$
60-84 Year olds	N=59	N=20	
Females	34	35	-
Current smokers	24	60	$p < 0.01$
Ex-smokers	20	5	-
Chronic cough	12	17	-
Recent Wheeze	22	5	-
Loose Cough	25	45	-
Rhonchi	10	15	-
Crepitations	27	15	-

* chi-square for the significant differences between the two groups.

9.4.3 Abnormal Lung Function

A total of 49 subjects had COPD and the mean FEV_1/FVC ratio in this group was 66% (SD 6.2). A total of 45 subjects had NOLD and the mean FEV_1/FVC ratio in this group was 91% (SD 6.9). Figure 9.1 shows the prevalence of ALF stratified by age and gender. Figures 9.2 and 9.3 show the prevalence of ALF stratified by age and community for males and females.

9.4.4 ALF in 7-19 and 20-39 year olds

Table 9.4 shows the prevalence of COPD and NOLD in 7-19 and 20-39 year olds stratified by community and gender. Tables 9.7 and 9.8 show the distribution of the "exploratory variables" for those with COPD, NOLD and normal lung function in these age groups. In 7-19 year olds sniffing petrol, recent wheeze and loose cough were significantly more prevalent among those with ALF. In 20-39 year olds BHR, asthma, recent wheeze, and crepitations were more significantly prevalent among those with ALF.

9.4.5 ALF in 40-59 and 60-84 year olds

Table 9.5 shows the prevalence of COPD and NOLD in 40-59 and 60-84 year olds stratified by community and gender. Tables 9.9 and 9.10 show the prevalence of the exploratory variables for these age groups. In 40-59 year olds ex-smokers, rhonchi and crepitations were more prevalent among those with ALF. In 60-84 year olds BHR, asthma and recent wheeze were more prevalent in those with ALF.

9.4.6 ALF in 20-84 year olds

Table 9.6 shows the prevalence of COPD and NOLD in 20-84 year olds stratified by community and gender. This table was included as a summary for adults. Table 9.11 shows the prevalence of the exploratory variables in this group by ALF status. In 20-84 year olds ex-smokers, BHR, asthma, recent wheeze, loose cough, rhonchi and crepitations were more prevalent among those with ALF. Current alcohol use was least prevalent among those with CLD.

9.4.7 Prevalence of COPD by Age, Gender and Region

Figure 9.4 shows the prevalence of COPD stratified by age and gender. Figures 9.5 and 9.6 show the prevalence of COPD stratified by age and community for males and females.

9.4.8 Adjusted Odds Ratios for CLD (ALF), COPD and NOLD

The adjusted odds ratios for CLD, COPD and NOLD are shown in table 9.12. Increasing age, ex-smoking, BHR and petrol sniffing were associated with increased odds for having CLD. A reduction in the odds for CLD was associated with living in CA3 .

Increasing age, ex-smoking and BHR were associated with an increase in the odds for having COPD. Living in CA3 was associated with a reduction in the odds. Gender was not an independent predictor for COPD.

Male gender and sniffing petrol were independent risk factors for NOLD. Age was not an independent predictor for this abnormality. Living in CY1 was independently associated with a significant reduction in the odds for having NOLD.

Table 9.4

Prevalence of Abnormal Lung Function in 7-19 year olds and 20-39 year olds by gender and community.

	N	% NOLD†	% COPD	% CLD	95% CI¤
Males 7-19					
CY1	66	1.5	0.0	1.5	0.0-4.4
CY2	20	0.0	0.0	0.0	0.0-14.5
CA3	85	5.9	0.0	5.9	0.9-10.9
CA4	39	23.1	0.0	23.1	9.8-36.3
All	**210**	**7.1**	**0.0**	**7.1**	**3.6-10.6**
Females 7-19					
CY1	68	0.0	0.0	0.0	0.0-5.3
CY2	14	14.3	0.0	14.3	0.0-32.6
CA3	61	1.6	0.0	1.6	0.0-4.7
CA4	43	2.3	0.0	2.3	0.0-6.8
All	**186**	**2.2**	**0.0**	**2.2**	**0.0-4.3**
Males 20-39					
CY1	57	0.0	8.8	8.8	1.5-16.2
CY2	18	0.0	0.0	0.0	0.0-15.0
CA3	46	6.5	4.3	10.8	1.8-19.8
CA4	31	19.4	0.0	19.4	5.5-33.3
All	**152**	**5.9**	**4.6**	**10.5**	**5.6-15.4**
Females 20-39					
CY1	91	1.1	1.1	2.2	0.0-5.2
CY2	27	3.7	0.0	3.7	0.0-10.8
CA3	63	0.0	4.8	4.8	0.0-10.1
CA4	48	4.2	0.0	4.2	0.0-9.9
All	**229**	**1.7**	**1.7**	**3.4**	**1.1-5.7**

† Non-obstructive lung disease.

¤ 95% confidence interval of the percentage with CLD.

Table 9.5

Prevalence of Abnormal Lung Function in 40-59 year olds and 60-84 year olds by gender and community.

	N	% NOLD†	% COPD	% CLD	95 % CI¤
Males 40-59					
CY1	31	0.0	29.0	29.0	13.1-44.9
CY2	9	0.0	22.2	22.2	0.0-49.3
CA3	24	4.2	8.3	12.5	0.0-25.7
CA4	24	33.3	12.5	45.8	0.0-65.7
All	**88**	**10.2**	**18.2**	**28.4**	**18.9-37.8**
Females 40-59					
CY1	41	2.4	19.5	21.9	9.2-34.5
CY2	10	0.0	20.0	20.0	0.0-44.8
CA3	29	0.0	0.0	0.0	0.0-11.1
CA4	23	0.0	4.3	4.3	0.0-12.6
All	**103**	**1.0**	**10.7**	**11.7**	**5.5-17.9**
Males 60-84					
CY1	20	5.0	40.0	45.0	23.2-66.8
CY2	2	0.0	0.0	0.0	0.0-51.0
CA3	8	0.0	12.5	12.5	0.0-35.4
CA4	9	22.2	11.1	33.3	2.5-64.1
All	**39**	**7.7**	**25.6**	**33.3**	**18.5-48.1**
Females 60-84					
CY1	3	0.0	0.0	0.0	0.0-43.9
CY2	-	-	-	-	-
CA3	11	0.0	0.0	0.0	0.0-27.7
CA4	6	0.0	16.7	16.7	0.0-46.5
All	**20**	**0.0**	**5.0**	**5.0**	**0.0-14.5**

† Non-obstructive lung disease.

¤ 95% confidence interval of the percentage with CLD.

Table 9.6

Prevalence of Abnormal Lung Function in 20-84 year olds by gender and community.

	N	% NOLD†	% COPD	% CLD	95 % CI¤
Males 20-84					
CY1	108	0.9	20.4	21.3	13.6-29.2
CY2	29	0.0	6.9	6.9	0.0-16.1
CA3	78	5.1	6.4	11.5	4.4-18.6
CA4	64	25.0	6.3	31.3	19.9-42.6
All	**279**	**7.5**	**11.8**	**19.3**	**14.8-23.9**
Females 20-84					
CY1	135	1.5	6.7	8.2	3.6-12.8
CY2	37	2.7	5.4	8.1	0.0-16.9
CA3	103	0.0	2.9	2.9	0.0-6.1
CA4	77	2.6	2.6	5.2	0.0-10.2
All	**352**	**1.4**	**4.5**	**5.9**	**3.4-8.4**

† Non-obstructive lung disease.

¤ 95% confidence interval of the percentage with CLD.

Table 9.7

Prevalence of exploratory variables (as a percentage) in 7-19 year olds by Abnormal Lung Function status.

	Normal	NOLD†	COPD	Chisq‡
N	377	19	0	
Current smoker	16.4	21.0	-	-
Ex-smoker	1.3	0.0	-	-
Ever-smoker	17.8	21.1	-	-
Petrol sniffer	4.8	26.3	-	$p < 0.001$
Alcohol drinker	13.5	26.3	-	-
BHR	0.8	0.0	-	-
Asthma	0.3	0.0	-	-
Chronic cough	6.8	11.1	-	-
Recent wheeze	3.5	15.8	-	$p < 0.01$
Loose cough	16.5	47.4	-	$p < 0.001$
Rhonchi	5.1	10.5	-	-
Crepitations	1.9	0.0	-	-

† Non-obstructive lung disease.

‡ The probability that the differences between the groups are significant using chi-square or Mantel-Haenszel chi-square.

Table 9.8

Prevalence of exploratory variables (as a percentage) in 20-39 year olds by Abnormal Lung Function status.

	Normal	NOLD†	COPD	Chisq‡
N	357	13	11	
Current smoker	52.9	61.5	54.6	-
Ex-smoker	7.8	7.7	9.1	-
Ever-smoker	60.8	69.2	63.6	-
Petrol sniffer	2.2	0.0	0.0	-
Alcohol drinker	57.1	61.5	54.6	-
BHR	2.8	23.1	36.4	$p < 0.001$
Asthma	1.1	7.7	18.2	$p < 0.001$
Chronic cough	8.2	7.7	9.1	-
Recent wheeze	7.0	7.7	27.3	$p < 0.05$
Loose cough	16.3	30.8	9.1	-
Rhonchi	2.5	7.7	0.0	-
Crepitations	3.1	23.1	9.1	$p < 0.01$

† Non-obstructive lung disease.

‡ The probability that the differences between the groups are significant using chi-square or Mantel-Haenszel chi-square.

Table 9.9

Prevalence of exploratory variables (as a percentage) in 40-59 year olds by Abnormal Lung Function status.

	Normal	NOLD†	COPD	Chisq‡
N	154	10	27	
Current smoker	40.3	30.0	48.2	-
Ex-smoker	13.0	30.0	29.6	$p < 0.05$
Ever-smoker	53.3	60.0	77.8	-
Petrol sniffer	0.0	0.0	0.0	-
Alcohol drinker	37.0	30.0	25.9	-
BHR	5.2	0.0	14.8	-
Asthma	2.0	0.0	3.7	-
Chronic cough	15.7	30.0	14.8	-
Recent wheeze	12.3	10.0	22.2	-
Loose cough	26.6	50.0	29.6	-
Rhonchi	7.1	0.0	22.2	$p < 0.05$
Crepitations	3.9	10.0	22.2	$p < 0.01$

† Non-obstructive lung disease.

‡ The probability that the differences between the groups are significant using chi-square or Mantel-Haenszel chi-square.

Table 9.10

Prevalence of exploratory variables (as a percentage) in 60-84 year olds by Abnormal Lung Function status.

	Normal	NOLD†	COPD	Chisq‡
N	45	3	11	
Current smoker	20.0	33.3	36.4	-
Ex-smoker	15.6	33.3	36.4	-
Ever-smoker	35.6	66.7	72.7	-
Petrol sniffer	0.0	0.0	0.0	-
Alcohol drinker	17.8	0.0	9.1	-
BHR	4.4	0.0	36.4	$p < 0.01$
Asthma	4.4	0.0	27.3	$p < 0.05$
Chronic cough	8.9	0.0	27.3	-
Recent wheeze	13.3	33.3	54.6	$p < 0.05$
Loose cough	24.4	66.7	18.2	-
Rhonchi	6.7	33.3	18.2	-
Crepitations	26.7	0.0	36.4	-

† Non-obstructive lung disease.

‡ The probability that the differences between the groups are significant using chi-square or Mantel-Haenszel chi-square.

Table 9.11

Prevalence of exploratory variables (as a percentage) in 20-84 year olds by Abnormal Lung Function status.

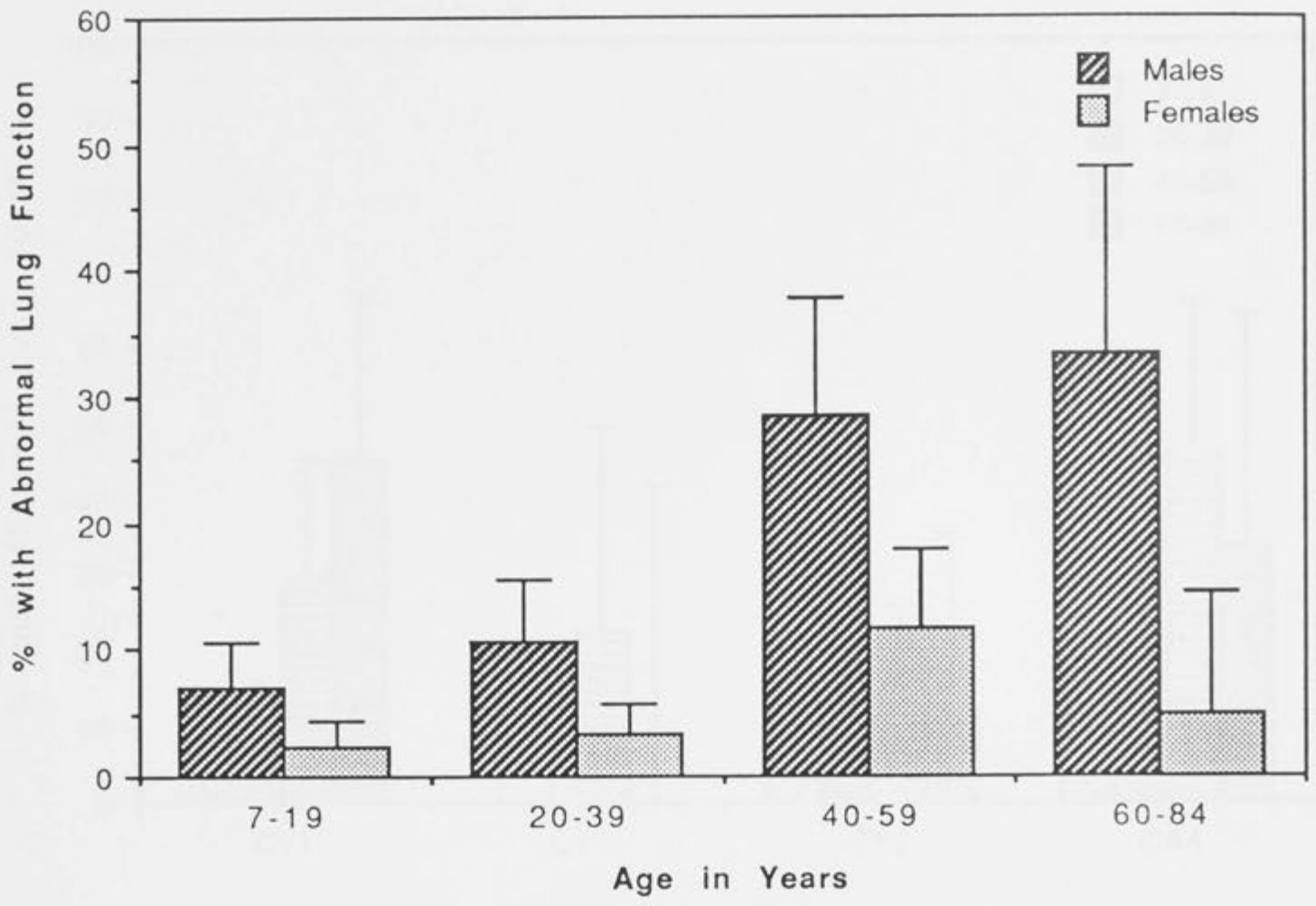
	Normal	NOLD†	COPD	Chisq‡
N	556	26	49	
Current smoker	46.8	46.2	46.9	-
Ex-smoker	9.9	19.2	26.5	$p < 0.005$
Ever-smoker	56.7	65.4	73.5	-
Petrol sniffer	1.4	0.0	0.0	-
Alcohol drinker	48.4	42.3	28.6	$p < 0.05$
BHR	3.6	11.5	24.5	$p < 0.001$
Asthma	1.6	3.9	12.2	$p < 0.001$
Chronic cough	10.3	15.4	16.3	-
Recent wheeze	9.0	11.5	30.6	$p < 0.001$
Loose cough	19.8	42.3	22.5	$p < 0.05$
Rhonchi	4.2	8.0	16.3	$p < 0.005$
Crepitations	5.2	15.4	22.5	$p < 0.001$

† Non-obstructive lung disease.

‡ The probability that the differences between the groups are significant using chi-square or Mantel-Haenszel chi-square.

Figure 9.1

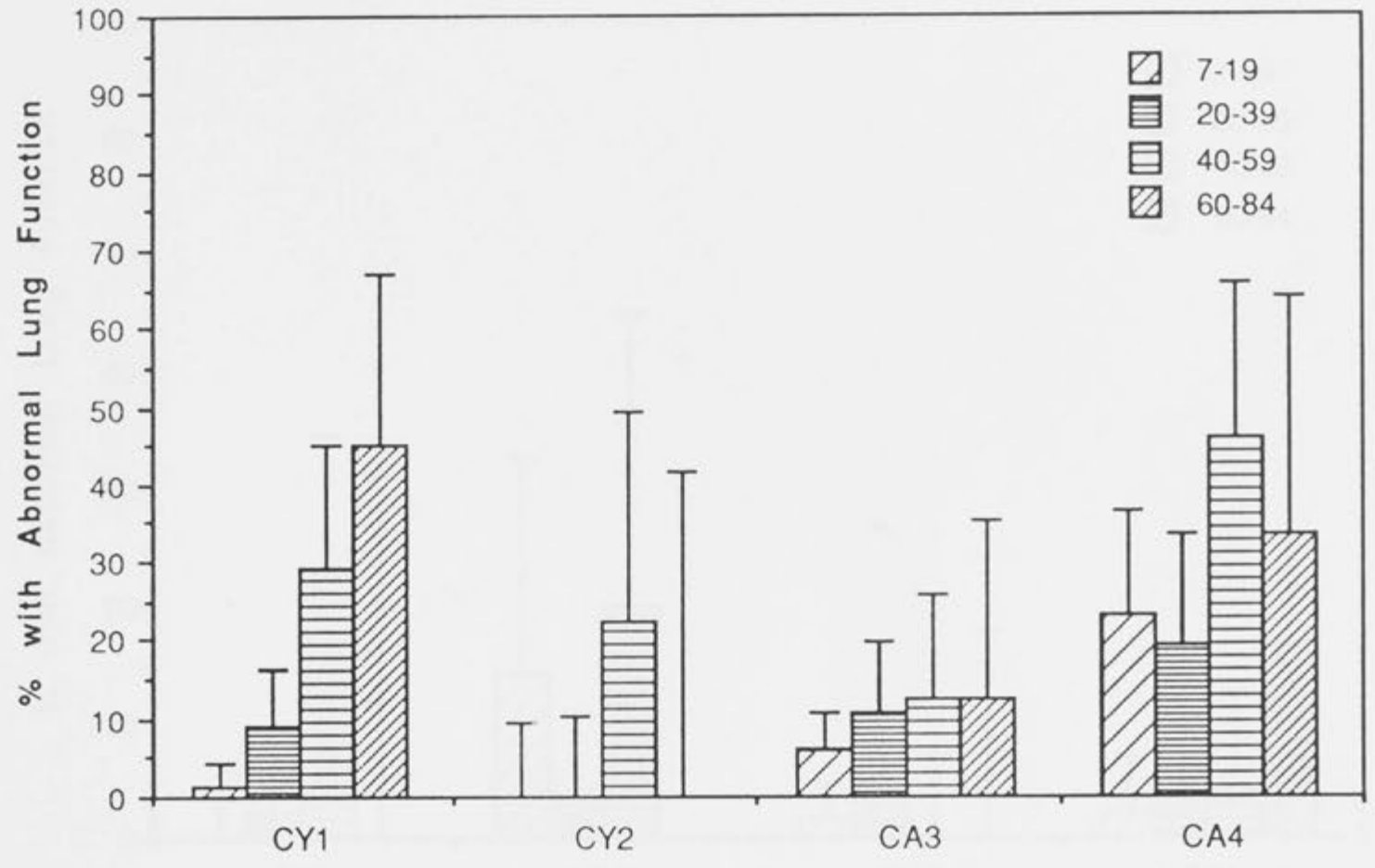
Prevalence of Chronic Lung Disease (ALF) by age and gender.



T bars indicate the upper limits of the 95% confidence interval of the estimates.

Figure 9.2

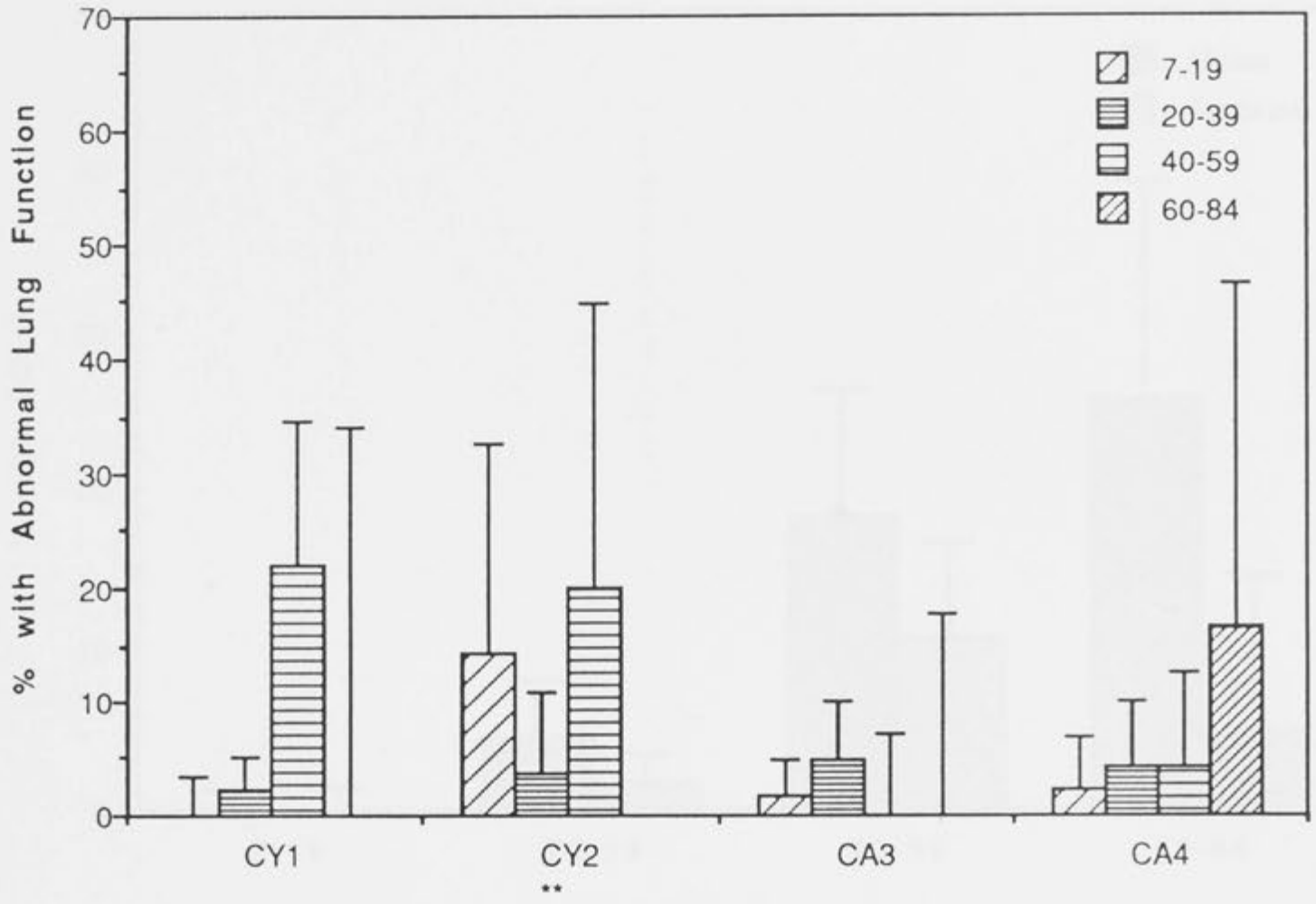
Prevalence of Chronic Lung Disease (ALF) in males by age and community.



T bars indicate the upper limits of the 95% confidence interval of the estimates.

Figure 9.3

Prevalence of Chronic Lung Disease (ALF) in females by age and community.

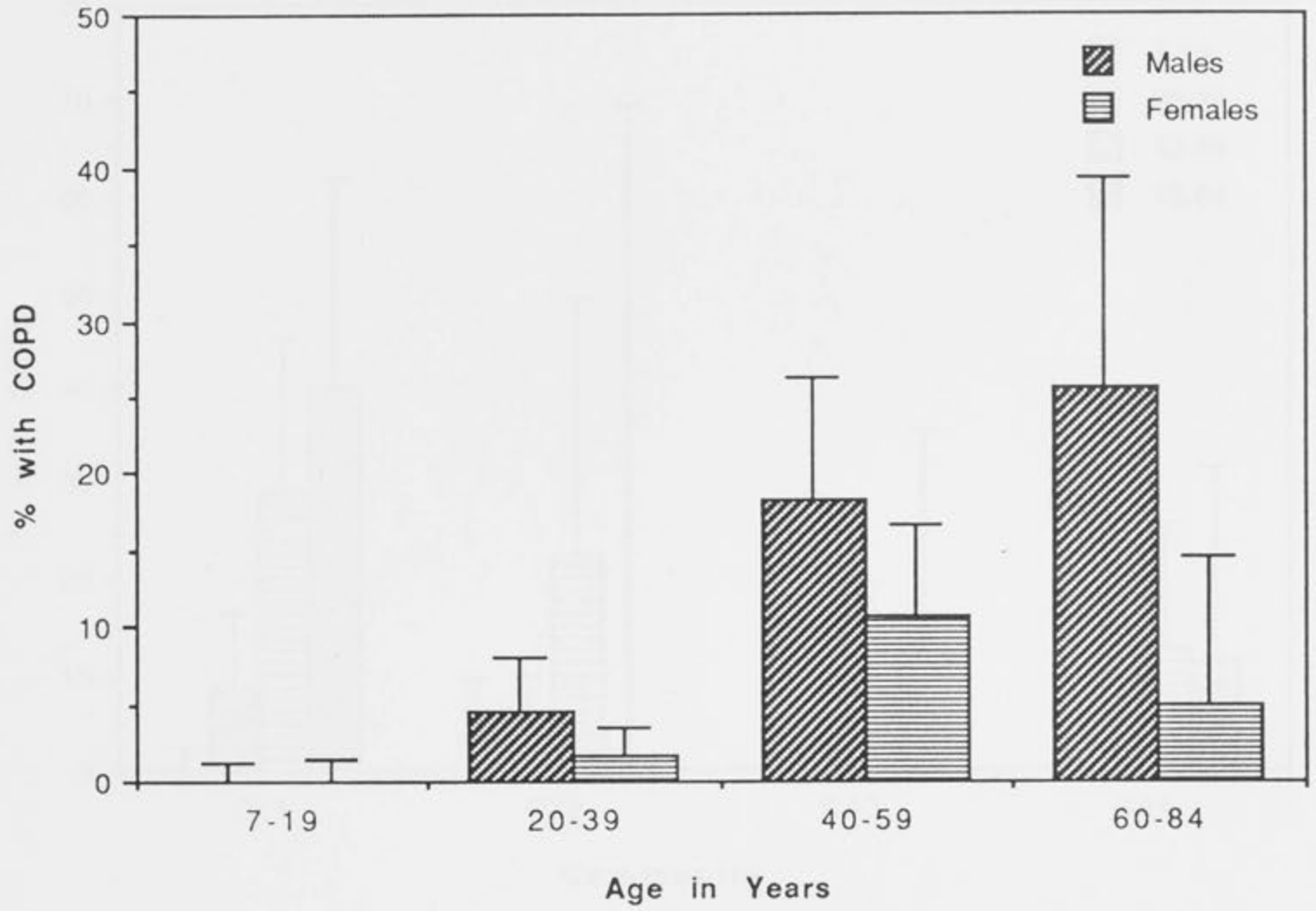


T bars indicate the upper limits of the 95% confidence interval of the estimates.

** There were no 60-84 year old women tested in CY2.

Figure 9.4

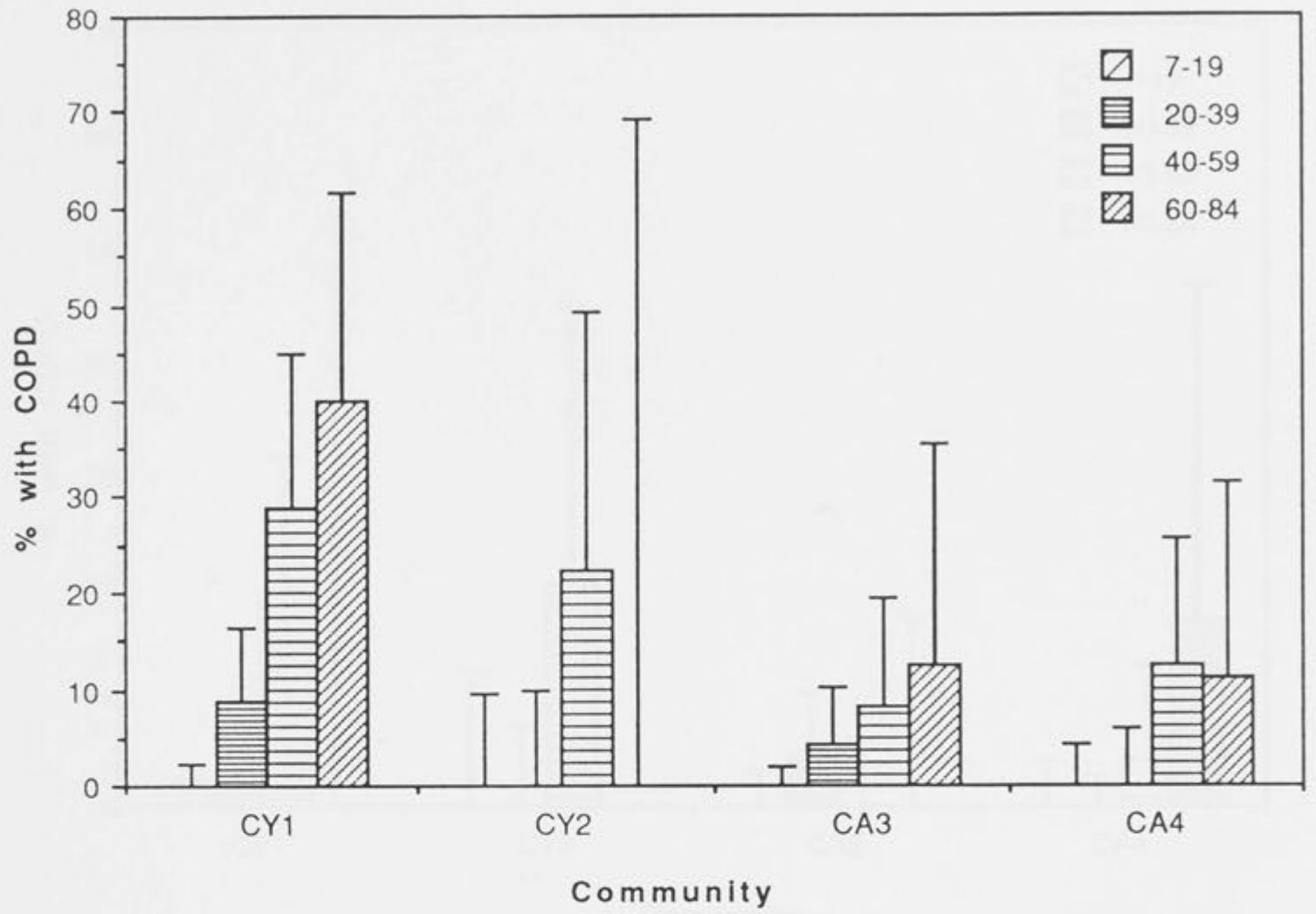
Prevalence of COPD by age and gender.



T bars indicate the upper limits of the 95% confidence interval of the estimates.

Figure 9.5

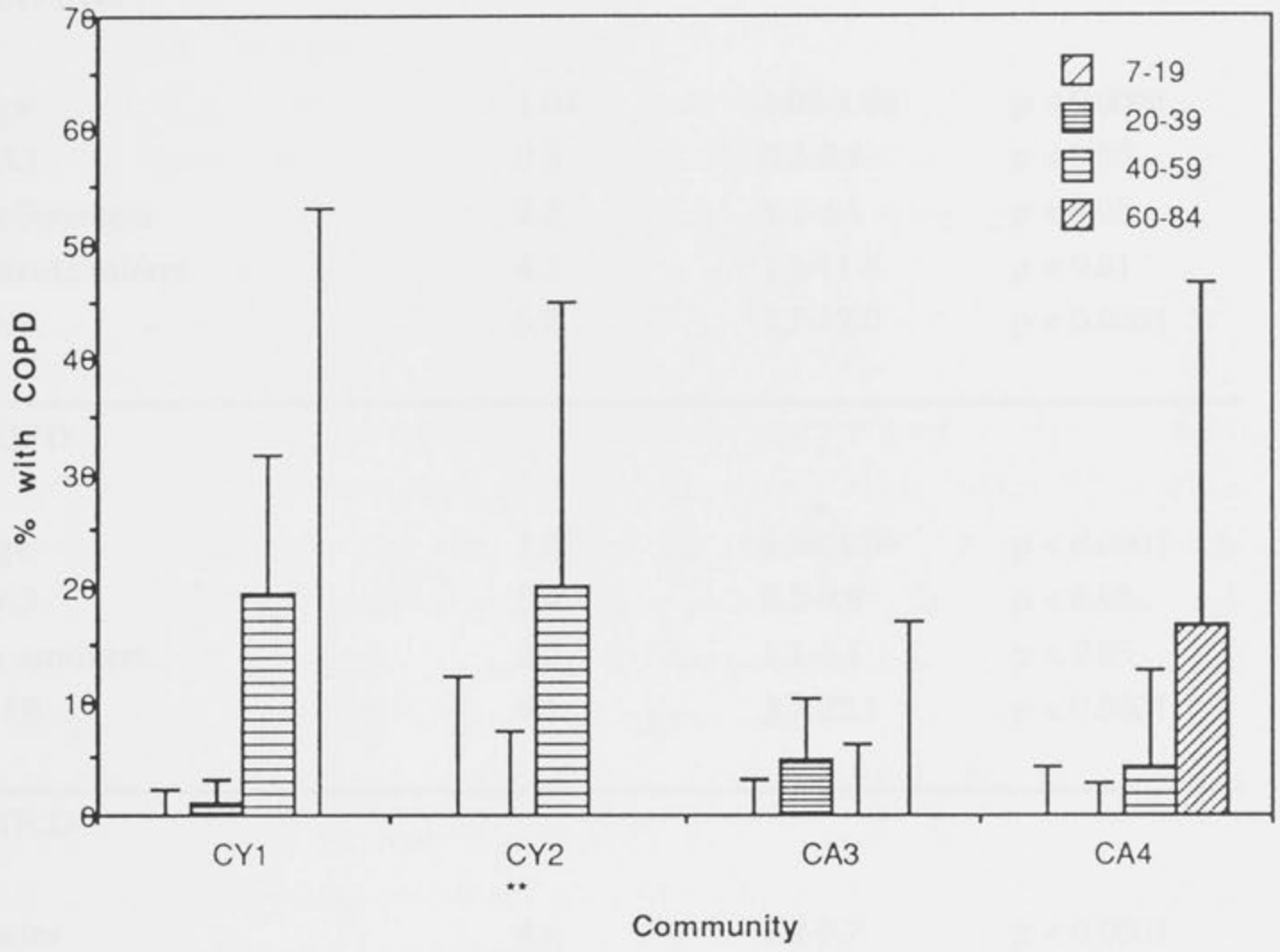
Prevalence of COPD in males by age and community.



T bars indicate the upper limits of the 95% confidence interval of the estimates

Figure 9.6

Prevalence of COPD in females by age and community.



T bars indicate the upper limits of the 95% confidence interval of the estimates.

** There were no 60-84 year old women tested in CY2.

Table 9.12

Adjusted odds ratios for Chronic Lung Disease, COPD and Non-obstructive Lung Disease (NOLD).

	Odds ratio	95% CI	p value
CLD (ALF)			
Age	1.04	1.03-1.06	$p < 0.0001$
CA3	0.5	0.3-0.9	$p < 0.05$
Ex-Smokers	2.2	1.1-4.1	$p < 0.05$
Petrol Sniffers	4.2	1.5-11.8	$p < 0.01$
BHR	5.7	2.7-12.0	$p < 0.0001$
COPD			
Age	1.07	1.05-1.09	$p < 0.0001$
CA3	0.4	0.2-0.9	$p < 0.05$
Ex-smokers	2.3	1.1-5.1	$p < 0.05$
BHR	9.1	3.7-22.1	$p < 0.0001$
NOLD			
Males	4.6	2.2-9.7	$p < 0.0001$
Petrol Sniffers	3.0	1.1-8.4	$p < 0.05$
CY1	0.17	0.06-0.49	$p < 0.001$

Odds ratio for males = the increase in odds for ALF in males.

Odds ratio for CY1 = the reduction in odds for ALF for subject resident in CY1.

Odds ratio for petrol sniffers = the increase in odds for ALF if subject sniffed petrol.

Odds ratio for age = the increase in odds for ALF with each incremental year of age.

Odds ratio for CA3 = the reduction in odds for ALF for subject resident in CA3.

Odds ratio for ex-smokers = increase in odds for ALF if subject was an ex-smoker.

Odds ratio for BHR = the increase in odds for ALF if $PD_{20}FEV_1$ was < 3.9 µmols histamine.

The odds ratios in the models are adjusted for confounding by each identified variable.

9.5 Discussion

The critical observations about CLD were that the western lifestyle of CY1 was associated with a high prevalence of COPD and a low prevalence of NOLD and that the lifestyle in CA3 and CA4 was associated with less COPD but a higher prevalence of NOLD. The multivariate analysis and the distribution of disease between the communities suggested that cigarette smoking was a risk factor for COPD and that petrol sniffing was a risk factor for NOLD. There was no evidence that current alcohol use was directly associated with CLD.

9.5.1 Technical Issues

In CY2 the high technically unsatisfactory proportion (and the relatively poor participation rate) precluded the drawing of firm conclusions about the prevalence of CLD in that community (see table 9.2). The relatively high rates of technically unsatisfactory spirometric tests in CY2 (and to a lesser extent in CY1) were due to embarrassment about performing the tests, particularly among adolescents, and failure to comprehend the instructions regarding the manoeuvres (eg taking in a full breath). Also they may have reflected the high prevalence of CLD because these data in table 9.3 show that a high percentage of 40-59 year old people with unsatisfactory spirometric tests were ex-smokers. Later in this chapter it is shown that ex-smokers were at increased risk of having CLD. Data from CY2 were included in this chapter for completeness. The proportions with technically unsatisfactory tests in CA3 and CA4 were low. It is unlikely that technical difficulties in the present study lead to over estimation of the prevalence of CLD.

Although subjects in the present study were divided into those with COPD and NOLD, some of the people with COPD may have had a mixed ventilatory defect with a low FEV_1 and FVC and abnormal FEV_1/FVC ratio. The author elected to ignore this distinction because further sub-division into other categories of CLD would have created smaller groups and analytical problems.

9.5.2 Prevalence of COPD

Table 9.4 shows that none of the 7-19 year old children had COPD. The overall prevalence of COPD among adults (see table 9.6) was high in comparison with most population-based studies performed in the United States of America (USA) where the prevalence of doctor diagnosed emphysema or ventilatory impairment (usually FEV_1 less than 60 or 65% predicted) has been 4 to 6% among adult men and 1 to 3% for women (45). Although the criteria for

defining COPD in these studies differed from those in the present study, the comparison suggests that the prevalence of COPD in the present study was high.

The increase in the prevalence of COPD with age (see figures 9.4 to 9.6) was consistent with the results of studies of CLD in Papua New Guinea (79) and COPD in the USA (45). The increase is probably a manifestation of accumulating exposure to risk factors for COPD with age.

9.5.3 Prevalence of NOLD

No comparable epidemiological studies of NOLD in other populations were located. Clinical experience suggests that the prevalence of NOLD in non-Aboriginal Australians is considerably less than the prevalence of COPD.

In men, the overall prevalence of NOLD was 7.1% in 7-19 year olds and 7.5 % in 20-84 year olds. This was probably higher than would be found if studies of NOLD were performed among non-Aboriginal Australians. One of the interesting features of NOLD was marked regional variation in the prevalence among males. The prevalence was very high in CA4 and low in CY1. In CA3 males the prevalence was higher than in CY1 but starkly less than that in CA4. The low prevalence of NOLD in CY1 and the reduction in odds for NOLD associated with living in that community (see table 9.12) suggests that life in this community conferred some protection against the development of this type of disease. This is discussed further in section 9.5.5 which examines the possible causes of NOLD

The overall prevalence of NOLD in females aged 7-19 years was 2.2% and among 20-84 year olds was 1.4%. Mirroring the finding in men, the prevalence of NOLD among women from CY1 was low. Although NOLD in females was significantly less prevalent than in males, the prevalence was probably higher than would be anticipated among non-Aboriginal Australians.

The prevalence of NOLD did not significantly increase with age in contrast to the prevalence of COPD. This suggests that the disease(s) that cause NOLD in the present study possibly had their genesis early in life rather than as a result of accumulating exposure to risk factors for lung disease.

9.5.4 The Associations and Clinical Features of COPD

The adjusted odds ratio for COPD in ex-smokers suggested that these people were 130% more likely to have COPD than current smokers or never-smokers (see table 9.12). This was circumstantial evidence that cigarette

smoking is an important risk factor for COPD and that subjects with COPD are more likely to give up smoking than those without this disease. The failure of current smoking to be directly associated with COPD may have reflected the dichotomous coding for smoking status, the low numbers of cigarettes consumed daily by some smokers (see chapter 5) and the verity that studies of non-Aboriginal people reveal only a minority of smokers eventually develop abnormal lung function (45). In addition it is recognised that the signal to noise ratio in cross-sectional studies* designed to measure the impact of smoking is high because of individual variation in lung function and confounding (111).

The second observation that suggested cigarette smoking was a risk factor for COPD was that the prevalence of COPD was highest in CY1 where the prevalence of smoking was high (68% in males and 56% in females) and conversely the prevalence of COPD was lowest among CA3 and CA4 females where smoking was least prevalent.

Although the conclusion that cigarette smoking was a cause of COPD has biological credibility (because it is a recognised cause of COPD in non-Aboriginal people), the present study design and numbers did not allow an assessment of the temporal relationship between cigarette smoking and COPD nor of dose response. Further research is required to examine longitudinally the impact of cigarette smoking on Aboriginal lung function and to determine if there are factors (eg NOLD in childhood) that put some smokers at particular risk for developing COPD.

The prevalence of BHR among those with COPD was significantly higher than in those without CLD (see tables 9.11 and 9.12). This partly reflected the finding that approximately 12% of those with COPD had asthma. However in 1985 Yan *et al* observed that the prevalence of BHR ($PD_{20}FEV_1$ less than 3.9 μmols of histamine) was 45% among non-asthmatic adults with respiratory symptoms and FEV_1/FVC ratios less than 70% (90). In the present study the prevalence of BHR among those with COPD was comparatively low.

BHR in non-Aboriginal people has been associated with cigarette smoking, airways inflammation and airflow obstruction but the inter-relationships

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* Longitudinal studies, by diminishing the noise of variation in lung function between individuals, are superior to cross-sectional studies for detecting the spirometric effects of smoking.

between these factors are complex and poorly understood (45). In the study by Yan *et al* cited above, 87% of the subjects with COPD were ever-smokers whereas in the present study the prevalence of ever-smokers among those with COPD was 73% (see table 9.11). This small difference in the prevalence of ever-smokers between these two studies was an unlikely explanation for the low prevalence of BHR among those with COPD in the present study. However the low prevalence of BHR may be an indication that the mix of pathological conditions that underlie COPD in the present study was different from the Yan *et al* study and that more subjects in the present study had disease that was not due to cigarette smoking (or asthma).

The low adjusted odds ratios for COPD (and CLD) for subjects from CA3 (see table 9.12 and figures 9.2, 9.3, 9.5 and 9.6) supported anecdotal reports of a low prevalence of CLD in that community (see chapter 3). The adjusted odds ratio showed that CA3 subjects were 50-60% less likely to have COPD (and CLD) than subjects in the other three communities. However the apparent protective effect was not strong and so could be secondary to sampling bias despite the high participation rate. Furthermore although the odds for COPD was reduced in this community, the prevalence of COPD was still at least as high as that found in the USA and 5-6% of men had NOLD. In conclusion it is tentatively concluded that the lifestyle in CA3 was associated with factors which confer some protection against the development of COPD.

The reasons for the apparently reduced odds for having COPD for people from CA3 are not known. The prevalence of COPD (2.9%) in CA3 women could be viewed as a baseline prevalence (without the influence of smoking) because only one woman smoked. The people of CA3 were probably exposed to similar amounts of environmental dust and smoke as people in other communities so it is unlikely that confounding by these agents reduced the odds for COPD in this community. It was noteworthy that in CA3 there was a difference in prevalence of both COPD and NOLD between men and women. As the men and women of CA3 shared the same environment, the most noticeable difference between the two groups was that 56% of men and 1% of women smoked. This could be interpreted as suggesting that either men in this community had some constitutional susceptibility to COPD* from factors other

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* Although in the present study the prevalence of COPD was higher in males than females, (see figure 9.1) the multivariate analysis did not show that male gender was an independent risk

than smoking, or as further evidence that smoking was an important cause of COPD in CA3. If smoking was an important cause of COPD in all the communities studied then the reduced odds for COPD in CA3 might be partially a product of confounding by regional variation in smoking pattern (see chapter 5). It is possible that the lifestyle in CA3 conferred protection against the development of COPD by fostering a cultural milieu which limits the use of substances that cause COPD.

In PNG COPD has been found to be more prevalent in women than men, to increase in prevalence with age and be unrelated to smoking home grown tobacco (brus) (79). In PNG domestic wood smoke pollution and lower respiratory tract infections in infancy have been considered aetiological possibilities for COPD but no firm conclusions have been reached regarding their roles. The findings in the present study confirm that factors other than cigarette smoking may cause COPD. Further research is required to identify these factors.

Table 9.11 shows that the symptoms and signs recorded in the present study had low sensitivities for detecting COPD. Only 20% of subjects reported chronic cough, 30% recent wheeze and less than 25% had loose cough or abnormalities of auscultation. These low sensitivities suggest that measurement spirometric function is necessary to detect COPD. The prevalence of chronic cough among those with COPD was lower than clinical experience might predict. The low prevalence may reflect the constraining criteria for a positive response to the question. The chronic cough question used in the present study appears to have little clinical utility.

9.5.5 The Associations and Clinical Features of NOLD

One of the remarkable findings about NOLD was the high prevalence in CA4 males (see table 9.4 and 9.6). The multivariate analysis revealed that male gender and sniffing petrol were significant risk factors for this disorder and that living in CA4 was not an independent risk factor (see table 9.12). Theoretically petrol sniffing could cause NOLD by promoting the development of pulmonary fibrosis or by significantly retarding the growth of FEV_1 and FVC during the adolescent years. The high prevalence of NOLD in the CA4 adults could be due to a high prevalence of people who had previously sniffed petrol. The strength of the relationship between petrol sniffing and NOLD and the

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factor. This suggested that males did not have any constitutional predisposition for developing COPD.

biological plausibility that this noxious substance might damage pulmonary tissue suggests that sniffing petrol is a risk factor for NOLD in Aboriginal communities. Although sniffing petrol was strongly associated with NOLD, only 26% of 7-19 year olds with NOLD were current sniffers suggesting that in this age group other aetiological factors were also important.

Bronchiectasis in Aboriginal communities has been associated with poor living conditions, early lower respiratory tract infections with concurrent dehydration, unspecified spirometric abnormalities and lung destruction (see chapter 2). In non-Aboriginal people with bronchiectasis, abnormalities of ventilatory function are commonly seen but no specific pattern of pulmonary dysfunction is recognised (114). Most patients with diffuse bronchiectasis have airflow obstruction but those with large amounts of atelectasis and fibrosis may have mixed obstructive/restrictive patterns or a largely restrictive pattern. Given that pneumonia is known to be prevalent and often recurrent in some Aboriginal communities (see chapter 2) it is possible that bronchiectasis (or some more subtle pathophysiological consequence of childhood pneumonia) could be an important cause of NOLD in the present study.

The prevalence and adjusted odds ratio for NOLD for people living in CY1 were low (see tables 9.4, 9.6 and 9.12). The magnitude of the reduction in the prevalence and the high participation rate in CY1 meant it was unlikely that sampling bias accounted for the finding. Because some NOLD was possibly a manifestation of pulmonary damage caused by early childhood pneumonia (eg bronchiectasis) the low prevalence of NOLD in CY1 may be secondary to the low prevalence of pneumonia*. The relatively good standard of housing in CY1 maybe the reason the prevalence of pneumonia in childhood was reported to be low in that community. Conversely it follows that the poorer standard of housing in CA3 and CA4 and the coexistent high incidence of lower respiratory tract infections in childhood might partially explain the higher prevalence of NOLD in those communities.

The inference from the present study that housing conditions influence the prevalence of pneumonia and NOLD together with the observation by Harris *et*

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* In chapter three it was noted that community health service staff had observed that pneumonia in childhood was uncommon in CY1 and CY2, common in CA3 (but rarely serious enough to cause evacuation) and common CA4 (and often serious enough to require evacuation to Alice Springs).

al (53) that the incidence of hospitalisation for pneumonia in Bourke children fell following housing improvements from 1968-69 to 1983-84 (see chapter 2) provide support for the notion that there is a causal link between living conditions for Aboriginal people and the prevalence of lung disease.

The prevalence of BHR among those with NOLD was low at 11.5%. None of the children with NOLD had BHR. Of the adults with NOLD, 3.5% had asthma. The findings regarding BHR in NOLD suggest that most of the people with this type of ventilatory impairment did not have associated airways inflammation.

Among the 7-19 year olds with NOLD, 11% had chronic cough, 16% had recent wheeze, 47% had loose cough, 11% had rhonchi and none had crepitations (see table 9.7). It is likely that the low prevalence of chronic cough reflected the stringent criteria that were required for a positive response in the questionnaire. The clinical findings in the adults with NOLD were similar to the 7-19 year olds although crepitations were heard in 15% of the subjects (see table 9.11). These findings suggest that respiratory symptoms and clinical signs are inadequate for identifying subjects with NOLD.

9.5.6 Synthesis and Diagnostic Possibilities for COPD

The findings discussed above suggest that some COPD was related to cigarette smoking and that 12% was due to asthma. COPD in non-Aboriginal smokers is believed to be secondary to small airways disease, emphysema or a combination of both. Whether COPD in Aborigines who smoke is due to similar pathological processes is unknown. Furthermore, little is known about the pathological basis of COPD in never-smokers. The clinical findings were of no practical clinical help in detecting or characterising the underlying abnormalities in those with COPD.

Some of the people with COPD in the present study may have had bronchiectasis because airflow obstruction is known to occur in people with this condition (114) and there are reasons to believe this condition may be prevalent (see chapter 2).

The role of environmental factors in the development of COPD in Aborigines remains unknown. The substantial number of never-smokers among those with COPD and the reduced odds for COPD amongst those from CA3 suggest that unidentified environmental factors may be important for the development

of COPD. Further research needs to focus on the risk factors and pathological basis for COPD in Aboriginal non-smokers.

9.5.7 Synthesis and Diagnostic Possibilities for NOLD

NOLD has been shown to be associated with petrol sniffing and the author contends that the present study supports the hypothesis that some NOLD is secondary to damage resulting from childhood pneumonia. Bronchiectasis, pulmonary fibrosis and impaired lung growth in childhood are possible pathological processes that could mediate these associations. A small proportion of adults with NOLD had asthma. The clinical findings were non-specific and therefore unhelpful in characterising the abnormalities present. Whether the prevalence of crepitations of 23% among 20-39 year olds with NOLD was an indication that this group had a high prevalence of pulmonary fibrosis is unknown. Further investigation of a group of subjects with NOLD and matched controls using CXRs, lung volume measurement and carbon monoxide diffusion capacity measurement would help characterise the abnormalities underlying this condition.

Further research needs to elucidate the factors which influence lung growth in childhood and adolescence, the impact of living conditions and the sequelae of childhood pneumonia.

9.5.7.1 Other possible risk factors for CLD

It is now recognised that maternal smoking during pregnancy may adversely effect foetal lung growth. This effect is independent of birth weight. Hanrahan *et al* studied the lung function of 80 healthy infants and found that the infants of non-smoking mothers had significantly better flow rates throughout the ventilatory cycle than infants born to smoking mothers (Am Rev Resp Dis 1992;145:1129-35). This finding was consistent with the hypothesis that maternal smoking impairs airway development *in utero*. In the present study it is possible that there were cohort effects on lung function due to the changing patterns of maternal smoking and that maternal smoking contributed to the high prevalence of CLD in some regions.

Secondly, it is now recognised that low birth weight may be associated with clinically significant abnormalities of ventilatory function that persist into adult life. In a retrospective cohort study of 5718 British men Barker *et al* showed that death from chronic obstructive airways disease was associated with low birth weight and weight at one year (BMJ 1991;303:671-75). In the report of the National Aboriginal Health Strategy Working Party of March 1989 it is noted that Aboriginal infants are more frequently born underweight than non-Aboriginal Australian children. It is possible that intrauterine growth retardation is a significant risk factor for CLD in Aboriginal communities.

Finally, although early respiratory tract infections may be a risk factor for the development of CLD such infections may actually be markers for children with diminished lung function. Barker *et al* showed that bronchitis or pneumonia in infancy (age < 1 year) was associated with a 0.17 litre reduction in adult FEV_1 independent of birth weight (and maternal smoking habit). Martinez *et al* have shown that diminished lung function is a risk factor for the subsequent development of lower respiratory tract infections. Therefore the role of early lower respiratory tract infections in the development of chronic lung disease remains controversial.

9.5.8 Clinical Implications of CLD

The present study has shown that spirometry is the only reliable method for detecting people with either COPD or NOLD. Funding should be made available to Aboriginal community health services to purchase spirometers* because it has been shown in this study that CLD is a significant public health problem. The performance of spirometric function in Aboriginal communities could lead to a wider recognition of the importance of CLD and could be used as a focus for health promotion.

Clinical orthodoxy encourages the early identification of people with CLD so that people at risk of developing more advanced lung disease can be counselled about risk factors for CLD and so appropriate therapy (eg for asthma) can be instituted. Therefore screening for CLD should probably become routine in Aboriginal health services. It has been recommended that all Aboriginal people with CLD receive pneumococcal immunisation to reduce the incidence of pneumococcal pneumonia (115) and people with CLD should also have an annual influenza immunisation (116). The findings in the present study suggest that approximately 20% of adult males and 5% of females would benefit from detection of their CLD and these vaccinations.

The high prevalence of COPD and NOLD was the major finding of this study. In non-Aboriginal people it has been shown that reduced ventilatory function is an independent risk factor for death from respiratory disease, ischaemic heart disease and other causes (117, 118). If the four study communities are similar to other rural Aboriginal communities in Australia, it is possible that high levels of COPD, NOLD and cigarette smoking explain a considerable proportion of the premature mortality suffered by the Aboriginal nation (27).

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* Provision would also need to be made for training health personnel in the use of the spirometers and interpretation of results.

9.6 Conclusion

- The overall prevalence of COPD in adult males was 11.8% and in adult females was 4.5%.
- COPD was not detected in children.
- Ex-smokers were at increased risk for having COPD.
- Increasing age was an independent risk factor for COPD.
- The lifestyle in CA3 was associated with a reduction in the likelihood of subjects having COPD.
- COPD was significantly associated with BHR however the prevalence of BHR among those with CLD was low in comparison with other studies.
- Approximately 12 % of those with COPD had asthma.
- The overall prevalence of NOLD was approximately 7% in males and 2% in females.
- Aboriginal people living in the community with the most adequately maintained houses had a reduced risk of developing NOLD.
- Petrol sniffing and male gender were independent risk factors for NOLD.
- Respiratory symptoms and signs had poor sensitivity for detecting CLD.
- Explanations other than petrol sniffing, cigarette smoking and asthma need to be invoked to explain the high prevalence of COPD and NOLD.
- Future research needs to focus on the factors which influence the growth and decline of lung function in Aborigines.

CHAPTER 10

Summary and Conclusion

10.1 Introduction

This chapter summarises the study's main findings and explores their research and public health and implications. There is an emphasis on ethical and process issues, the importance of smoking, the prevalence of asthma and atopy, the characteristics of normal lung function and the nature and distribution of chronic lung disease.

10.2 Ethics, Consultation and Feedback

This study showed that the National Health and Medical Research Council's guidelines for health research in Aboriginal and Torres Strait Islander communities are practical and facilitate community cooperation (3). Their implementation was labour and time intensive but resulted in a process that was orientated towards community needs. The author's willingness to consult and adapt to meet community concerns and the involvement of community members were crucial to the study's success.

The commitment to appropriate feedback was welcomed. Early feedback was valued as was the creation of permanent reports for inclusion in medical records. In the communities where English literacy was low, personal copies of written reports were of less interest. With time, interest shifted from a focus on individual results to an emphasis on the collective implications. Community elders accorded high priority to discussions with children about health issues emerging from the research.

10.3 Cigarette Smoking

The study confirmed that cigarette smoking is an important public health problem for Aboriginal people. Aborigines have a high prevalence of

diabetes, hypertension and obesity, so smoking places them at great risk from cardiovascular as well as respiratory disease (64).

The prevalence of smoking varied by gender and region. In Cape York the prevalence was approximately 70%. In central Australia smoking was less prevalent but probably increasing among women. The most frequently reported age for commencing smoking was 13 years. There was regional variation in the number of cigarettes smoked daily. Self-reported cigarette use was highest in Cape York. If the uptake of smoking in teenagers cannot be reduced in central Australia the prevalence of smoking may rise to that in Cape York.

There was a strong association between cigarette smoking and alcohol consumption. This linkage suggests that initiatives to encourage smoking cessation should consider the context in which it occurs.

Knowledge of the health consequences of smoking was limited. This finding suggested that most people were not in a position to make informed decisions about smoking. In one community this knowledge was associated with a reduction in the odds for being a current smoker. This observation raises the possibility that initiatives to increase knowledge would reduce the prevalence of smoking. It seems likely that adequately resourced Aboriginal health services could develop educational programs and strategies that would discourage the commencement of smoking and help smokers quit.

10.4 Asthma and Atopy

Childhood asthma was almost non-existent and among adults the prevalence was low in comparison with that found in non-Aboriginal Australians. There was significant regional variation in the prevalence of atopy and asthma. The only risk factor identified for asthma was allergy to cats. The prevalence of asthma in the Cape York children was low despite exposure to levels of *Der p*1 allergen sufficient to cause sensitisation and wheezing in non-Aboriginal children. In CY1 there was no evidence that asthma or atopy were associated with non-Aboriginal HLA DR and DQ alleles. The prevalence of BHR among adults was similar to that found among non-Aboriginal Australians but in children the prevalence was relatively low. The risk factors for BHR were HDM allergy, feline allergy and cigarette smoking. The prevalence of atopy among adults was similar to that found among non-Aboriginal Australian adults. However in children it was significantly lower

than that found among their non-Aboriginal peers so it seems that the Aboriginal children have delayed atopy acquisition.

The asthma findings are interesting because asthma is said to be a commonly observed clinical problem among urban Aboriginal children. Environmental factors might be crucial for the expression of asthma in Aborigines. It needs to be determined whether asthma in urban Aboriginal children is associated with an atopy acquisition rate that is higher than that observed in this rural study.

10.5 Normal Lung Function

The values predicted for FEV_1 among healthy people in the present study were approximately 20% lower than those predicted for Caucasians of the same height, age and gender and for FVC they were 30% lower. The FEV_1/FVC ratio was consequently high in comparison with Caucasians. Asymptomatic cigarette smokers had higher predicted values of FEV_1 and FVC than non-smokers. The rate of decline of FEV_1 and FVC with age in healthy smokers and non-smokers appeared comparable to that observed in Caucasians.

The spirometric function reference value equations developed in the present study are the first complementary set for Aboriginal adults and children. Although it is recognised that black Americans and other indigenous people also have low spirometric values in comparison with Caucasians, the values predicted for the asymptomatic Aborigines in this study were even lower. This suggested that either Aboriginal people have a particular genetic predisposition to comparatively low spirometric values or that most of the population have compromised lung function. The finding that asymptomatic smokers had better lung function than non-smokers could be secondary to a health selection effect because it has been observed that children previously hospitalised for respiratory illness are less likely to commence smoking than children without such a history (5).

The equations developed during the present study will allow informed assessment of individual lung function in rural Aborigines. This will benefit practitioners who assess respiratory problems and health services that screen for respiratory disease. Also epidemiologists now have a tool to identify CLD. More research is required to investigate the determinants of lung growth and decline in Aborigines.

10.6 Chronic Lung Disease

The prevalence of CLD was high in comparison with that found among non-Aboriginal Australians. Respiratory symptoms and signs were not useful for identifying or characterising subjects with chronic obstructive pulmonary disease (COPD) or non obstructive lung disease (NOLD).

The prevalence of COPD was 11.8% in males and 4.5% in females. COPD was not detected in children. Ex-smokers were at increased risk of having COPD. Increasing age was an independent risk factor for COPD. The traditional lifestyle in CA3 was associated with an independent reduction in the risk of having COPD but confounding by smoking pattern may have influenced the result. Twelve percent of subjects with COPD had asthma. BHR was associated with COPD but the prevalence of BHR among those with COPD was low compared with that found in non-Aboriginal people with this disease.

The prevalence of NOLD was 7% in males and 2% in females. Petrol sniffing and male gender were significant independent risk factors for NOLD. Age was not an independent risk factor for this abnormality. Subjects from the community with the most adequate houses (CY1) were significantly less likely to have NOLD than subjects from communities where the housing was of a poor standard and where serious lower respiratory tract infections in childhood are said to be common.

The findings suggest that initiatives that lead to more Aboriginal people living in decentralised family groups in adequately maintained houses on their land have the potential to reduce the burden of COPD and NOLD. The mechanisms by which these factors reduce the prevalence of CLD need further investigation.

The high prevalence of COPD and NOLD in the present study probably explains why respiratory disease is an important cause of hospitalisation and death among Aborigines. Furthermore, the findings may have other implications for mortality because in non-Aboriginal people, impaired lung function is also a risk factor for death from cardiovascular disease and other disorders. Thus the significance of CLD as a cause of death among Aboriginal people has probably been under-estimated in the past.

The low prevalence of symptoms amongst those with CLD means that CLD can only be reliably detected with a spirometer. The present study findings suggest that all Aboriginal medical services should be equipped with spirometers so

that people can be screened for CLD. All people with CLD should be counselled about smoking and petrol sniffing (when relevant) and they should also be offered pneumococcal and influenza immunisation (115).

Further research which explores the factors which affect the growth and decline of lung function in Aborigines is needed. Many children may be failing to achieve their full spirometric potential by the end of adolescence. Because it is often asymptomatic and yet a major cause of premature Aboriginal death, community awareness regarding the importance of CLD needs to be enhanced.

10.7 Conclusion

Cigarette smoking, petrol sniffing, COPD and NOLD are major public health problems for Australian Aborigines. Knowledge of the health consequences of smoking is poor. Asthma is present in 3-5% of adults but it is almost non-existent in children. Aboriginal people have lower predicted spirometric values based on age, gender and height compared with other indigenous people and Caucasians.

The "traditional" lifestyle of CA3 was associated with significantly less COPD than the lifestyle in the three less traditional communities. The reasons for this are yet to be identified but possibly the lifestyle has fostered circumstances that have limited substance abuse. There was no evidence that the lifestyle in CA3 protected individuals from NOLD.

The lifestyle in the community where housing was most adequate (CY1) was associated with significantly less NOLD than the lifestyle in the communities where housing was less adequate. The low prevalence of respiratory tract infections that are reported to accompany the provision of adequate housing are a biologically plausible mechanism by which adequate housing could protect people from developing NOLD.

This study lends support to the contention that decentralised living in family groups yields health advantages for Aborigines, that initiatives to improve Aboriginal housing will lead to a fall in the prevalence of NOLD, and that appropriate smoking education programs are imperative. The report of the national Aboriginal health strategy working party (28) offers a policy which, if implemented, would make healthy lifestyle options, culturally appropriate health education services and adequate housing available to more Aborigines. This study reinforces the need for the full implementation of that strategy.

References

1. Thompson JE, Sleigh AC, Passey ME, Barnes AB, Streatfield RW. Ventilatory standards for clinically well Aboriginal adults. Med J Aust 1992;156:566-569.

2. Henderson H. Recommendations from the workshop 'Preventing smoking-related diseases among Aboriginals'. Aboriginal Health Information Bulletin 1990 13-15.

3. National Health and Medical Research Council. Guidelines on ethical matters in Aboriginal and Torres Strait Islander health research. Commonwealth of Australia, 1991.

4. Hanna J, Torzillo P. Acute respiratory infections in Australian Aboriginal children: current knowledge and future requirements. PNG Med J 1991;34:204-210.

5. Gold DR, Tager IB, Weiss ST, Tosteson TD, Speizer FE. Acute lower respiratory illness in childhood as a predictor of lung function and chronic respiratory symptoms. Am Rev Respir Dis 1989;140:877-884.

6. Maxwell GM. Chronic chest disease in Australian Aboriginal children. Arch Dis Child 1972;47:897-901.

7. Lowry S. Housing and health. Br Med J 1989;299:1261-1262.

8. McCarthy P, Byrne D, Harrisson S, Keithley J. Respiratory conditions: effect of housing and other factors. J Epidemiol Community Health 1985;39:15-19.

9. Golding J. Child health and the environment. Br Med Bull 1986;42(2):204-211.

10. Heppell M. Aboriginal camps and housing: A black reality. Canberra: Australian Institute of Aboriginal Studies, 1979:250.

11. Nganampa Health Council Inc., South Australian Health Commission, Aboriginal Health Organisation of South Australia. Uwankara palyankyu kanyintjaku: An environmental and public heath review within the Anangu Pitjantjatjara lands. 1987.

12. Schenker MB, Samet JM, Speizer FE. Risk factors for childhood respiratory disease. Am Rev Respir Dis 1983;128:1036-1043.

13. Strope GL, Stempel DA. Risk factors associated with the development of chronic lung disease in children. Pediatr Clin North Am 1984;31(4):757-771.

14. Crapo J, Miller FJ, Mossman B, Pryor WA, Kiley JP. NHLBI Workshop Summary: Environmental Lung Diseases. Am Rev Respir Dis 1992;145:1506-1512.

15. Guest C, O'Dea K, Carlin J, Larkins R. Smoking in Aborigines and persons of European descent in southeastern Australia: prevalence and associations with food habits, body fat distribution and other cardiovascular risk factors. Aust J Public Health 1992;16(4):397-402.

16. Stephenson J. Tobacco smoking by Aboriginal people in Wilcania, Western N.S.W. The Health Promotion Unit of the Orana and Far West Region of the N.S.W. Department of Health, 1990.

17. Watson C, Fleming J, Alexander K. A survey of drug use patterns in Northern Territory Aboriginal communities: 1986-1987. Northern Territory Department of Health and Community Services, Drug and Alcohol Bureau, 1988.

18. O'Donnell TV. Asthma—Australia and New Zealand. Aust NZ J Med 1988;18:303-309.

19. Redshaw GM. Letter to the protector of Islanders, Thursday Island: Re Health and sanitation at Lockhart River mission. Commonwealth of Australia, Department of Health and Quarantine Service, 1940.

20. Kamien M. The physical health of Aboriginal children in Bourke, New South Wales. Med J Aust 1976;1:33-37.

21. Moodie PM. Aboriginal health. Canberra: Australian National University Press, 1973

22. Gandevia B. The prevalence of signs of chronic respiratory disease in Pintubi and Walbiri Aborigines at Papunya, central Australia, and Warburton, Western Australia. Med J Aust 1967;2(6):237-42.

23. Torzillo PJ, Waterford JE, Hollows FC, Jones DL. Respiratory disease amongst Aborigines in the Pilbara. Int J Epidemiol 1983;12(1):105-6.

24. Devanesen D, Furber M, Hampton D, Honari M, Kinmonth N, Peach HD. Health indicators in the Northern Territory. Northern Territory Department of Health, Darwin, 1986.

25. Yusuf F, Hamilton A. Hospital morbidity among Aboriginal Australians in New South Wales. J Biosoc Sci 1982;14:445-463.

26. Chandler D, Dugdale A. Respiratory function in Aboriginal school children. Med J Aust 1979;1(Spec. Suppl.):10-12.

27. Plant AJ. Aboriginal mortality in the Northern Territory 1979-83 [Master of Public Health]. University of Sydney, 1988.

28. National Aboriginal Health Strategy Working Party. A national Aboriginal health strategy. 1989.

29. Sporik R, Holgate ST, Platts-Mills TA, Cogswell JJ. Exposure to house-dust mite allergen (Der P1) and the development of Asthma in childhood. N Engl J Med 1990;323(8):502-507.

30. Yan K, Salome C, Woolcock AJ. Rapid method for measurement of bronchial responsiveness. Thorax 1983;38:760-765.

31. Brady M. Psychoactive substance use among young Aboriginal Australians. Federal Legal Publications, Inc, 1991

32. Urapuntja Health Service. Health survey (Compiled by Gault A). Institute for Aboriginal Development, 1990.

33. Hicks DG. Aboriginal mortality rates in Western Australia 1983. Health Department of Western Australia, Perth, 1985.

34. Gray A, ed. Causes of Aboriginal mortality: A matter of life and death. Canberra: Aboriginal Studies Press, 1990:

35. Maxwell GM, Elliott RB, McCoy WT, Langsford WA. Respiratory infections in Australian Aboriginal children: A clinical and radiological study. Med J Aust 1968;(Nov 17):990-993.

36. Maxwell GM. Chronic respiratory disease in Aboriginal children. First Australian Ross Conference. Cronulla N.S.W: Ross Laboratories, 1972: 31-37.

37. Maxwell GM. Rarity of mucoviscidosis in Australian Aborigines. Lancet 1971;(June 5):1190.

38. Peat JK, van-der-Berg RH, Mellis CM, Leeder SR, Woolcock AJ. Changes in the prevalence of asthma and allergy in Australian children (Abstract). Thoracic Society of Australia and New Zealand annual scientific meeting. Sydney, 1993: 01.

39. Hiller HG. A year's survey of paediatric chest X-rays in the Alice Springs hospital. Aust Radiol 1975;19:32-35.

40. Dawson KP, Long A, Kennedy J, Mogridge N. The chest radiograph in acute bronchiolitis. J. Paediatr. Child Health 1990;26:209-211.

41. Hiller. The radiological follow-up of Aboriginal children with bronchiectasis treated surgically or medically. Aust Paediatric J 1976;12:319-320.

42. Bateson E, Woo-Ming M. Destroyed lung. A report of cases in West Indians and Australian Aborigines. Clin Radiol 1976;27:223-226.

43. Bateson EM. Destroyed lung in the Australian Aboriginal. Aust Radiol 1978;22:117-124.

44. Kamien M. The physical health of Aboriginal adults in Bourke, New South Wales. Med J Aust 1976;1:38-44.

45. Hensley MJ, Saunders NA, ed. Clinical epidemiology of chronic obstructive pulmonary disease. New York/Basel: Dekker, 1989:Lung Biology in Health and Disease; vol 43).

46. Peat JK, Haby M, Spijker J, Berry G, Woolcock AJ. Prevalence of asthma in adults in Busselton, Western Australia. Br Med J 1992;305:1326-1329.

47. Chandler D, Dugdale A. Respiratory function tests in Aboriginal children on Mornington Island. Med J Aust 1980;1(Spec. Suppl.):10.

48. American Thoracic Society. Lung function testing: Selection of reference values and interpretative strategies. Am Rev Respir Dis 1991;144:1202-1218.

49. Harris MF. Early childhood pneumonia in Aborigines of Bourke, New South Wales. Med J Aust 1984;705-707.

50. Clark EH, Vincenc K, Tolley S. Skin atopy and bronchial hyperreactivity in full blood Aboriginal children. The Thoracic Society of Australia annual scientific meeting. Australia, 1984: 545.

51. Harris LA, Lane AR, Steele RH, Henderson TR. Preliminary report on the structure and function of nasal cilia in Aboriginal children of the Kimberley region of WA [abstract]. Aust Paediatric J 1984;20(3):242.

52. Watson DS, Watson RM, Siskind V. Respiratory function in Aboriginal children. Med J Aust 1986;144:S11-13.

53. Harris MF, Kamien M. Change in Aboriginal childhood morbidity and mortality in Bourke 1971-84. J. Paediatr. Child Health 1990;26:80-84.

54. Nganampa Health Council. Health report 1985/86. 1987.

55. Australian Bureau of Statistics. Aboriginal people in the Northern Territory. Australian Bureau of Statistics, 1990:103.

56. Hill D, Gray N. Australian patterns of tobacco smoking and related health beliefs. Community Health Stud 1984;V111(3):307-316.

57. Lake P. Alcohol and cigarette use by urban Aboriginal people. Aboriginal Health Information Bulletin 1989 May 1989:20-22.

58. Berndt RM, Berndt CH. The world of the first Australians. Sydney: Landsdown Press, 1981

59. Thompson D. Notes on the smoking-pipes of North Queensland and the Northern Territory of Australia. Man 1939;39:81-91.

60. Daube M. Proceedings of the pre-conference workshop "Preventing smoking related diseases among Aboriginals". Perth Western Australia: Seventh World Conference on Tobacco and Health, 1990: 20.

61. Henderson H. Unpublished study. Health Department of Western Australia 1989.

62. Taylor JC. Of acts and axes [Doctor of Philosophy]. James Cook University, 1984.

63. Jarvis M, Belcher M, Vesey C, Hutchison D. Low cost carbon monoxide monitors in smoking assessment. Thorax 1986;41:886-887.

64. Doll R, Peto R. Mortality in relation to smoking : 20 years' observations on male British doctors. Br Med J 1976;2:1525-1536.

65. Turner KJ, Stewart GA, Woolcock AJ, Green W, Alpers MP. Relationship between mite densities and the prevalence of asthma: comparative studies in two populations in the Eastern Highlands of Papua New Guinea. Clin Allergy 1988;18:331-340.

66. Platts-Mills TA, de Weck AL. Dust mite allergens and asthma: A world wide problem. J Allergy Clin Immunol 1989;83(2 pt 1):416-427.

67. Green WF, Sedgwick C, Woolcock AJ, Leeder SR, Stuckey M. House dust mites and skin tests in different Australian localities. Aust NZ J Med 1986;16:639-643.

68. Marsh DG, Meyers DA, Bias WB. The epidemiology and genetics of atopic allergy. N Engl J Med 1981;305:1551-1559.

69. Britton WJ, Woolcock AJ, Peat JK, Sedgwick CJ, Lloyd DM, Leeder SR. Prevalence of bronchial hyperresponsiveness in children: The relationship between asthma and skin reactivity to allergens in two communities. Int J Epidemiol 1986;15(No 2):202-209.

70. Du-Toit JI, Woolcock AJ, Salome CM, Sundrum R, Black JL. Characteristics of bronchial hyperresponsiveness in smokers with chronic air-flow limitation. Am Rev Respir Dis 1986;134:498-501.

71. Toelle BG, Peat JK, Salome CM, Mellis CM, Woolcock AJ. Toward a definition of asthma for epidemiology. Am Rev Respir Dis 1992;146:633-637.

72. Pepys J. Skin testing. Br J Hosp Med 1975;14:412-17.

73. Gao X, Veale A, Serjeantson S. AB1: A novel HLA-DPB1 allele found in one third of an Australian population. Immunogenetics 1992;36:64-66.

74. Hennekens CH, Buring JE. Epidemiology in medicine. (1st ed.) Boston: Little, Brown and Company, 1987:383.

75. Sackett DL, Haynes RB, Guyatt GH, Tugwell P. Clinical epidemiology: A basic science for clinical medicine. (Second ed.) Boston/Toronto/London: Little, Brown and Company, 1991

76. Landis JR, Koch GG. The measurement of observer agreement for categorical data. Biometrics 1977;33:159-74.

77. Dowse GK, Turner KJ, Woolcock AJ, Alpers MP. Emerging asthma in the Okapa district of the Eastern Highlands province of Papua New Guinea: The problem and its implications. PNG Med J 1983;26(1).

78. Warrell DA, Fawcett IW, Harrison BD, *et al* . Bronchial asthma in the Nigerian Savanna region. Q J Med 1975;44:325-47.

79. Attenborough RD, Alpers MA, ed. Human Biology in Papua New Guinea: The small cosmos. 1st ed. Melbourne: Clarendon Press, 1992:

80. Arlian LG, Vyszenski-Moher DL, Ahmed SG, Estes SA. Cross-antigenicity between the scabies mite, Sarcoptes scabiei, and the house dust mite, Dermatophagoides pteronyssinus. J Invest Dermatol 1991;96:349-354.

81. Jones HI. Intestinal parasite infections in Western Australian Aborigines. Med J Aust 1980;2:375-380.

82. Carswell F, Merrett J, Merrett TG, Meakins RH, Harland PS. IgE, parasites and asthma in Tanzanian children. Clin Allergy 1977;7:445-453.

83. Donnelly PM, Yang TS, Peat JK, Woolcock AJ. What factors explain racial differences in lung volumes? Eur Respir J 1991;4:829-838.

84. Rossiter CE, Weill H. Ethnic differences in lung function: evidence for proportional differences. Int J Epidemiol 1974;3(No 1):55-61.

85. Seltzer CC, Siegelaub AB, Friedman GD, Collen MF. Differences in pulmonary function related to smoking habits and race. Am Rev Respir Dis 1974;110:598-608.

86. Hsi BP, Hsu KH, Jenkins DE. Ventilatory functions of normal children and young adults: Mexican-American, white, and black: (3) Sitting height as a predictor. J Pediatr 1983;102(6):860-865.

87. Ware JH, Dockery DW, Louis TA, Xu X, Ferris B, Speizer FE. Longitudinal and cross-sectional estimates of pulmonary function decline in never smoking adults. Am J Epidemiol 1990;132(No. 4):685-700.

88. Burrows B, Knudson R, Lebowitz M. A descriptive analysis of the growth and decline of lung function. Chest 1983;5:717-724.

89. Sherrill D, Camilli A, Lebowitz M. On the temporal relationships between lung function and somatic growth. Am Rev Respir Dis 1989;140:638-644.

90. Yan K, Salome CM, Woolcock AJ. Prevalence and nature of bronchial hyperresponsiveness in subjects with chronic obstructive pulmonary disease. Am Rev Respir Dis 1985;132:25-29.

91. American Thoracic Society. Standardisation of spirometry—1987 update. Am Rev Respir Dis 1987;136:1285-1298.

92. SAS Institute Inc. SAS/STAT Users guide version 6. (Fourth ed.) Cary: SAS Institute Inc., 1989

93. Morris JF, Koski A, Johnson L. Spirometric standards for healthy nonsmoking adults. Am Rev Respir Dis 1971;103:57-67.

94. Vedal S, Schenker MB, Samet JM, Speizer FE. Risk factors for childhood respiratory disease. Am Rev Respir Dis 1984;130:187-192.

95. Crapo RO, Morris AH, Gardner RM. Reference spirometric values using techniques and equipment that meet ATS recommendations. Am Rev Respir Dis 1981;123:659-664.

96. Irwin R, Curley F, French C. Chronic cough: The spectrum and frequency of causes, key components of the diagnostic evaluation, and outcome of specific therapy. Am Rev Respir Dis 1990;141:640-647.

97. Jaakkola MS, Jaakkola JJ, Ernst P, Becklake MR. Respiratory symptoms in young adults should not be overlooked. Am Rev Respir Dis 1993;147:359-366.

98. Hall GJ, Gandevia B. Relationship of loose cough sign to daily sputum volume. Brit J Prev Soc Med 1971;25:109-13.

99. Armitage P, Berry G. Statistical methods in medical research. (2nd ed.) Oxford: Blackwell Scientific Publication, 1987

100. Concato J, Feinstein AR, Holford TR. The risk of determining risk with multivariable models. Ann Intern Med 1993;118:201-210.

101. Clifford RD, Radford M, Howell JB, Holgate ST. Prevalence of respiratory symptoms among 7 and 11 year old school children and association with asthma. Arch Dis Child 1989;64:1118-25.

102. Cullen KJ. Respiratory disease in a rural community. Lancet 1968;(Sept 21):657-660.

103. Whithey CW, Price CE, Swan AV, Papacosta AO, Hensley MJ. Repeatability of a questionnaire to assess respiratory symptoms in smokers. J Epidemiol Community Health 1988;42:54-9.

104. Samet JM, Tager IB, Speizer FE. The relationship between respiratory illness in childhood and chronic air-flow obstruction in adulthood. Am Rev Respir Dis 1983;127:508-523.

105. Beck GJ, Doyle CA, Schachter EN. A longitudinal study of respiratory health in a rural community. Am Rev Respir Dis 1982;125:375-381.

106. Tager IB, Munoz A, Rosner B, Weiss ST, Carey V, Speizer FE. Effect of cigarette smoking on the pulmonary function of children and adolescents. Am Rev Respir Dis 1985;131:752-759.

107. Lebowitz MD. The trends in airway obstructive disease morbidity in the Tucson epidemiological study. Am Rev Respir Dis 1989;140:S35-S41.

108. Morris K, Morganlander M, Coulehan J, Gahagen S, Arena V. Wood-burning stoves and lower respiratory tract infection in American Indian children. Am J Dis Child 1990;144:105-108.

109. Samet JM, Marbury MC, Spengler JD. Health effects and sources of indoor air pollution. Am Rev Respir Dis 1987;136:1486-1508.

110. Crapo J, Miller F, Mossman B, Pryor W, Kiley J. Relationship between acute inflammatory responses to air pollutants and chronic lung disease. Am Rev Respir Dis 1992;145:1506-1512.

111. Becklake MR. Occupational exposures: Evidence for a causal association with chronic obstructive pulmonary disease. Am Rev Respir Dis 1989;140:S85-S91.

112. Speizer FE. The rise in chronic obstructive pulmonary disease mortality: Overview and summary. Am Rev Respir Dis 1989;140:S106-S107.

113. Kellie SE, Attfield MD, Hankinson JL, Castellan RM. Spirometry variability criteria-association with respiratory morbidity and mortality in a cohort of coal miners. Am J Epidemiol 1987;125:437-444.

114. Fishman AP. Pulmonary diseases and disorders. New York: McGraw-Hill Book Company, 1988; vol 2).

115. Torzillo PJ. Pneumococcal vaccine: current status. Aust NZ J Med 1993;23:285-290.

116. Douglas RG. Prophylaxis and treatment of Influenza. N Engl J Med 1990;322:443-450.

117. Moon-Bang K, Gergen P, Kramer R, Cohen B. The effect of pulmonary impairment on all-cause mortality in a national cohort. Chest 1993;103:536-40.

118. Tockman MS, Comstock GW. Respiratory risk factors and mortality: Longitudinal studies in Washington County, Maryland. Am Rev Respir Dis 1989;140:S56-S63.

Appendices

Erratum

The footnote for appendices 6-17 should read:

L95 = lower limit of the reference range (5th percentile).

Appendix 1

Prevalence of atopy (as a percentage) to DP, DF, cockroach and dust by community and age.

	CY1	CY2	CA3	CA4	All
Number					
5-7	51	17	28	23	119
8-12	95	28	58	31	212
13-19	54	16	86	50	206
20-84	278	102	186	149	715
5-84	**478**	**163**	**358**	**253**	**1252**
DP					
5-7	10	0	4	22	10
8-12	16	18	10	16	15
13-19	24	25	17	24	21
20-84	31	30	16	27	26
5-84	**25**	**24**	**14**	**25**	**22**
DF					
5-7	10	0	4	13	8
8-12	14	7	5	19	11
13-19	17	12	9	24	15
20-84	25	26	11	21	21
5-84	**20**	**19**	**9**	**20**	**17**
Cockroach					
5-7	8	0	7	4	6
8-12	14	7	12	10	12
13-19	24	12	14	10	15
20-84	23	16	11	11	17
5-84	**20**	**13**	**12**	**10**	**15**
Dust					
5-7	2	0	0	0	1
8-12	5	4	2	0	3
13-19	5	0	2	2	3
20-84	10	7	2	5	6
5-84	8	5	2	3	5

Appendix 2

Prevalence of atopy (as a percentage) to cat, plantain, rye grass and *Alternaria* by community and age.

	CY1	CY2	CA3	CA4	All
Number					
5-7	51	17	28	23	119
8-12	95	28	58	31	212
13-19	54	16	86	50	206
20-84	278	102	186	149	715
5-84	**478**	**163**	**358**	**253**	**1252**
Cat					
5-7	0	0	0	4	1
8-12	1	0	0	0	0
13-19	5	0	0	0	1
20-84	8	1	0	2	3
5-84	**5**	**1**	**0**	**2**	**2**
Plantain					
5-7	0	0	0	0	0
8-12	0	0	0	0	0
13-19	2	0	0	0	0
20-84	2	1	0	2	1
5-84	**1**	**1**	**0**	**1**	**1**
Rye Grass					
5-7	0	0	0	0	0
8-12	1	0	2	0	1
13-19	2	0	1	2	1
20-84	5	2	2	2	3
5-84	**4**	**1**	**1**	**2**	**2**
Alternaria					
5-7	0	0	0	0	0
8-12	1	0	0	0	0
13-19	2	0	1	0	1
20-84	2	1	0	1	1
5-84	**1**	**1**	**0**	**1**	**1**

Appendix 3

Prevalence of atopy (as a percentage) to dog, horse, feathers and ragweed by community and age.

	CY1	CY2	CA3	CA4	All
Number					
5-7	51	17	28	23	119
8-12	95	28	58	31	212
13-19	54	16	86	50	206
20-84	278	102	186	149	715
5-84	478	163	358	253	1252
Dog					
5-7	0	0	N/A	N/A	0
8-12	1	0	N/A	N/A	1
13-19	2	0	N/A	N/A	0
20-84	4	2	N/A	N/A	3
5-84	2	1	N/A	N/A	2
Horse					
5-7	2	0	N/A	N/A	1
8-12	0	0	N/A	N/A	0
13-19	0	0	N/A	N/A	0
20-84	2	1	N/A	N/A	2
5-84	1	1	N/A	N/A	1
Feathers					
5-7	0	0	N/A	N/A	0
8-12	0	0	N/A	N/A	0
13-19	0	0	N/A	N/A	0
20-84	0	0	N/A	N/A	0
5-84	0	0	N/A	N/A	0
Ragweed					
5-7	0	0	N/A	N/A	0
8-12	0	0	N/A	N/A	0
13-19	0	6	N/A	N/A	1
20-84	2	1	N/A	N/A	2
5-84	1	1	N/A	N/A	1

Appendix 4

Prevalence of atopy (as a percentage) to Timothy grass, *Aspergillus*, acacia and bottlebrush by community and age.

	CY1	CY2	CA3	CA4	All
Number					
5-7	51	17	28	23	119
8-12	95	28	58	31	212
13-19	54	16	86	50	206
20-84	278	102	186	149	715
5-84	478	163	358	253	1252
Timothy Grass					
5-7	0	0	N/A	N/A	0
8-12	1	0	N/A	N/A	1
13-19	2	0	N/A	N/A	1
20-84	3	1	N/A	N/A	3
5-84	2	1	N/A	N/A	2
Aspergillus					
5-7	0	0	N/A	N/A	0
8-12	0	0	N/A	N/A	0
13-19	0	0	N/A	N/A	0
20-84	3	2	N/A	N/A	3
5-84	2	1	N/A	N/A	2
Acacia					
5-7	N/A	N/A	0	4	2
8-12	N/A	N/A	3	3	3
13-19	N/A	N/A	0	6	2
20-84	N/A	N/A	2	3	2
5-84	N/A	N/A	1	4	2
Bottle Brush					
5-7	N/A	N/A	4	0	2
8-12	N/A	N/A	2	3	2
13-19	N/A	N/A	0	4	1
20-84	N/A	N/A	1	3	2
5-84	N/A	N/A	1	3	2

Appendix 5

Prevalence of atopy (as a percentage) to eucalyptus, melaleuca and pine (Australian) by community and age.

	CY1	CY2	CA3	CA4	All
Number					
5-7	51	17	28	23	119
8-12	95	28	58	31	212
13-19	54	16	86	50	206
20-84	278	102	186	149	715
5-84	478	163	358	253	1252
Eucalyptus					
5-7	N/A	N/A	0	0	0
8-12	N/A	N/A	0	0	0
13-19	N/A	N/A	0	0	0
20-84	N/A	N/A	0	3	1
5-84	N/A	N/A	0	2	1
Melaleuca					
5-7	N/A	N/A	4	4	4
8-12	N/A	N/A	2	0	1
13-19	N/A	N/A	0	4	1
20-84	N/A	N/A	0	3	2
5-84	N/A	N/A	1	3	2
Pine					
5-7	N/A	N/A	0	0	0
8-12	N/A	N/A	0	0	0
13-19	N/A	N/A	0	0	0
20-84	N/A	N/A	0	1	1
5-84	N/A	N/A	0	1	0

Appendix 6

FEV_1 in litres for Aboriginal males aged 9-19 years.

	Age										
Ht	9	10	11	12	13	14	15	16	17	18	19
110	0.71	0.78	0.86	0.93	1.00	1.08	1.15	1.22	1.29	1.37	1.44
L95	0.03	0.10	0.17	0.24	0.31	0.37	0.43	0.50	0.56	0.62	0.68
115	0.86	0.94	1.01	1.08	1.16	1.23	1.30	1.38	1.45	1.52	1.60
L95	0.19	0.26	0.33	0.40	0.47	0.53	0.60	0.66	0.73	0.79	0.85
120	1.02	1.09	1.16	1.24	1.31	1.38	1.46	1.53	1.60	1.68	1.75
L95	0.35	0.42	0.49	0.56	0.63	0.70	0.76	0.83	0.89	0.95	1.01
125	1.17	1.25	1.32	1.39	1.47	1.54	1.61	1.69	1.76	1.83	1.91
L95	0.51	0.58	0.65	0.72	0.79	0.86	0.92	0.99	1.05	1.12	1.18
130	1.33	1.40	1.47	1.55	1.62	1.69	1.77	1.84	1.91	1.99	2.06
L95	0.66	0.74	0.81	0.88	0.95	1.02	1.09	1.15	1.22	1.28	1.34
135	1.48	1.56	1.63	1.70	1.78	1.85	1.92	2.00	2.07	2.14	2.22
L95	0.82	0.89	0.97	1.04	1.11	1.18	1.25	1.31	1.38	1.44	1.51
140	1.64	1.71	1.78	1.86	1.93	2.00	2.08	2.15	2.22	2.30	2.37
L95	0.97	1.05	1.12	1.20	1.27	1.34	1.41	1.47	1.54	1.61	1.67
145	1.79	1.87	1.94	2.01	2.08	2.16	2.23	2.30	2.38	2.45	2.52
L95	1.13	1.20	1.28	1.35	1.42	1.49	1.56	1.63	1.70	1.77	1.83
150	1.95	2.02	2.09	2.17	2.24	2.31	2.39	2.46	2.53	2.61	2.68
L95	1.28	1.36	1.43	1.51	1.58	1.65	1.72	1.79	1.86	1.93	1.99
155	2.10	2.18	2.25	2.32	2.39	2.47	2.54	2.61	2.69	2.76	2.83
L95	1.43	1.51	1.58	1.66	1.73	1.81	1.88	1.95	2.02	2.09	2.15
160	2.26	2.33	2.40	2.48	2.55	2.62	2.70	2.77	2.84	2.92	2.99
L95	1.58	1.66	1.74	1.81	1.89	1.96	2.03	2.11	2.18	2.24	2.31
165	2.41	2.48	2.56	2.63	2.70	2.78	2.85	2.92	3.00	3.07	3.14
L95	1.73	1.81	1.89	1.97	2.04	2.12	2.19	2.26	2.33	2.40	2.47
170	2.57	2.64	2.71	2.79	2.86	2.93	3.01	3.08	3.15	3.23	3.30
L95	1.88	1.96	2.04	2.12	2.19	2.27	2.34	2.42	2.49	2.56	2.63
175	2.72	2.79	2.87	2.94	3.01	3.09	3.16	3.23	3.31	3.38	3.45
L95	2.03	2.11	2.19	2.27	2.34	2.42	2.50	2.57	2.64	2.71	2.78
180	2.88	2.95	3.02	3.10	3.17	3.24	3.32	3.39	3.46	3.53	3.61
L95	2.17	2.26	2.34	2.42	2.49	2.57	2.65	2.72	2.80	2.87	2.94
185	3.03	3.10	3.18	3.25	3.32	3.40	3.47	3.54	3.62	3.69	3.76
L95	2.32	2.40	2.48	2.56	2.64	2.72	2.80	2.87	2.95	3.02	3.09

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 7

FVC in litres for Aboriginal males aged 9-19 years.

	Age										
Ht	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**
110	0.79	0.88	0.96	1.04	1.13	1.21	1.30	1.38	1.47	1.55	1.64
L95	0.03	0.12	0.19	0.27	0.35	0.43	0.50	0.57	0.65	0.72	0.79
115	0.95	1.04	1.12	1.21	1.29	1.37	1.46	1.54	1.63	1.71	1.80
L95	0.20	0.29	0.36	0.44	0.52	0.60	0.67	0.75	0.82	0.89	0.96
120	1.11	1.20	1.28	1.37	1.45	1.54	1.62	1.70	1.79	1.87	1.96
L95	0.36	0.45	0.53	0.61	0.69	0.77	0.84	0.92	0.99	1.06	1.13
125	1.27	1.36	1.44	1.53	1.61	1.70	1.78	1.86	1.95	2.03	2.12
L95	0.53	0.61	0.69	0.78	0.86	0.93	1.01	1.09	1.16	1.23	1.31
130	1.43	1.52	1.60	1.69	1.77	1.86	1.94	2.03	2.11	2.19	2.28
L95	0.69	0.78	0.86	0.94	1.02	1.10	1.18	1.26	1.33	1.40	1.48
135	1.59	1.68	1.76	1.85	1.93	2.02	2.10	2.19	2.27	2.36	2.44
L95	0.85	0.94	1.02	1.11	1.19	1.27	1.35	1.42	1.50	1.57	1.65
140	1.76	1.84	1.92	2.01	2.09	2.18	2.26	2.35	2.43	2.52	2.60
L95	1.01	1.10	1.18	1.27	1.35	1.43	1.51	1.59	1.67	1.74	1.82
145	1.92	2.00	2.09	2.17	2.25	2.34	2.42	2.51	2.59	2.68	2.76
L95	1.17	1.26	1.35	1.43	1.51	1.60	1.68	1.76	1.83	1.91	1.99
150	2.08	2.16	2.25	2.33	2.42	2.50	2.58	2.67	2.75	2.84	2.92
L95	1.33	1.42	1.51	1.59	1.68	1.76	1.84	1.92	2.00	2.08	2.15
155	2.24	2.32	2.41	2.49	2.58	2.66	2.74	2.83	2.91	3.00	3.08
L95	1.49	1.58	1.66	1.75	1.84	1.92	2.00	2.09	2.17	2.24	2.32
160	2.40	2.48	2.57	2.65	2.74	2.82	2.91	2.99	3.07	3.16	3.24
L95	1.64	1.73	1.82	1.91	2.00	2.08	2.17	2.25	2.33	2.41	2.49
165	2.56	2.64	2.73	2.81	2.90	2.98	3.07	3.15	3.24	3.32	3.40
L95	1.80	1.89	1.98	2.07	2.16	2.24	2.33	2.41	2.49	2.57	2.65
170	2.72	2.80	2.89	2.97	3.06	3.14	3.23	3.31	3.40	3.48	3.57
L95	1.95	2.04	2.13	2.22	2.31	2.40	2.49	2.57	2.65	2.74	2.82
175	2.88	2.97	3.05	3.13	3.22	3.30	3.39	3.47	3.56	3.64	3.73
L95	2.10	2.20	2.29	2.38	2.47	2.56	2.64	2.73	2.81	2.90	2.98
180	3.04	3.13	3.21	3.30	3.38	3.46	3.55	3.63	3.72	3.80	3.89
L95	2.26	2.35	2.44	2.53	2.63	2.71	2.80	2.89	2.97	3.06	3.14
185	3.20	3.29	3.37	3.46	3.54	3.62	3.71	3.79	3.88	3.96	4.05
L95	2.41	2.50	2.60	2.69	2.78	2.87	2.96	3.05	3.13	3.22	3.30

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 8

FEV_1 in litres for Aboriginal male non-smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
160	2.77	2.67	2.56	2.45	2.35	2.24	2.13	2.03	1.92	1.81	1.71
L95	1.93	1.82	1.72	1.61	1.51	1.40	1.29	1.19	1.08	0.97	0.86
165	2.98	2.88	2.77	2.66	2.56	2.45	2.34	2.24	2.13	2.02	1.92
L95	2.14	2.04	1.93	1.83	1.72	1.61	1.51	1.40	1.29	1.18	1.07
170	3.19	3.08	2.98	2.87	2.76	2.66	2.55	2.44	2.34	2.23	2.12
L95	2.35	2.25	2.14	2.04	1.93	1.82	1.72	1.61	1.50	1.39	1.28
175	3.40	3.29	3.19	3.08	2.97	2.87	2.76	2.65	2.55	2.44	2.33
L95	2.56	2.46	2.35	2.24	2.14	2.03	1.92	1.82	1.71	1.60	1.49
180	3.61	3.50	3.40	3.29	3.18	3.08	2.97	2.86	2.76	2.65	2.54
L95	2.77	2.66	2.56	2.45	2.35	2.24	2.13	2.02	1.92	1.81	1.70
185	3.82	3.71	3.60	3.50	3.39	3.28	3.18	3.07	2.96	2.86	2.75
L95	2.97	2.87	2.76	2.66	2.55	2.44	2.34	2.23	2.12	2.01	1.90
190	4.03	3.92	3.81	3.71	3.60	3.49	3.39	3.28	3.17	3.07	2.96
L95	3.18	3.07	2.97	2.86	2.75	2.65	2.54	2.43	2.33	2.22	2.11
195	4.23	4.13	4.02	3.91	3.81	3.70	3.59	3.49	3.38	3.27	3.17
L95	3.38	3.27	3.17	3.06	2.96	2.85	2.74	2.64	2.53	2.42	2.31
200	4.44	4.34	4.23	4.12	4.02	3.91	3.80	3.70	3.59	3.48	3.38
L95	3.58	3.48	3.37	3.26	3.16	3.05	2.94	2.84	2.73	2.62	2.51

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 9

FVC in litres for Aboriginal male non-smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
160	2.95	2.85	2.76	2.66	2.56	2.46	2.36	2.27	2.17	2.07	1.97
L95	1.93	1.84	1.74	1.64	1.55	1.45	1.35	1.25	1.15	1.05	0.95
165	3.22	3.12	3.02	2.92	2.83	2.73	2.63	2.53	2.43	2.34	2.24
L95	2.20	2.11	2.01	1.91	1.82	1.72	1.62	1.52	1.42	1.32	1.22
170	3.48	3.39	3.29	3.19	3.09	2.99	2.90	2.80	2.70	2.60	2.50
L95	2.47	2.38	2.28	2.18	2.08	1.99	1.89	1.79	1.69	1.59	1.49
175	3.75	3.65	3.55	3.46	3.36	3.26	3.16	3.06	2.97	2.87	2.77
L95	2.74	2.64	2.54	2.45	2.35	2.25	2.15	2.05	1.95	1.85	1.75
180	4.02	3.92	3.82	3.72	3.62	3.53	3.43	3.33	3.23	3.13	3.04
L95	3.00	2.91	2.81	2.71	2.61	2.52	2.42	2.32	2.22	2.12	2.02
185	4.28	4.18	4.09	3.99	3.89	3.79	3.69	3.60	3.50	3.40	3.30
L95	3.26	3.17	3.07	2.97	2.88	2.78	2.68	2.58	2.48	2.38	2.28
190	4.55	4.45	4.35	4.25	4.16	4.06	3.96	3.86	3.76	3.66	3.57
L95	3.52	3.43	3.33	3.23	3.14	3.04	2.94	2.84	2.74	2.64	2.54
195	4.81	4.72	4.62	4.52	4.42	4.32	4.22	4.13	4.03	3.93	3.83
L95	3.78	3.69	3.59	3.49	3.39	3.30	3.20	3.10	3.00	2.90	2.80
200	5.08	4.98	4.88	4.78	4.69	4.59	4.49	4.39	4.29	4.20	4.10
L95	4.04	3.94	3.85	3.75	3.65	3.55	3.45	3.36	3.26	3.16	3.06

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 10

FEV_1 in litres for Aboriginal male smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
160	2.91	2.81	2.70	2.59	2.49	2.38	2.27	2.17	2.06	1.95	1.85
L95	2.07	1.96	1.86	1.75	1.64	1.54	1.43	1.32	1.21	1.10	0.99
165	3.12	3.01	2.91	2.80	2.69	2.59	2.48	2.37	2.27	2.16	2.05
L95	2.28	2.18	2.07	1.96	1.86	1.75	1.64	1.53	1.42	1.32	1.21
170	3.33	3.22	3.12	3.01	2.90	2.80	2.69	2.58	2.48	2.37	2.26
L95	2.49	2.39	2.28	2.17	2.07	1.96	1.85	1.74	1.64	1.53	1.42
175	3.54	3.43	3.32	3.22	3.11	3.00	2.90	2.79	2.68	2.58	2.47
L95	2.70	2.60	2.49	2.38	2.28	2.17	2.06	1.95	1.84	1.74	1.63
180	3.75	3.64	3.53	3.43	3.32	3.21	3.11	3.00	2.89	2.79	2.68
L95	2.91	2.80	2.70	2.59	2.48	2.38	2.27	2.16	2.05	1.94	1.83
185	3.95	3.85	3.74	3.63	3.53	3.42	3.31	3.21	3.10	2.99	2.89
L95	3.12	3.01	2.90	2.80	2.69	2.58	2.47	2.37	2.26	2.15	2.04
190	4.16	4.06	3.95	3.84	3.74	3.63	3.52	3.42	3.31	3.20	3.10
L95	3.32	3.21	3.11	3.00	2.89	2.79	2.68	2.57	2.46	2.35	2.24
195	4.37	4.27	4.16	4.05	3.95	3.84	3.73	3.63	3.52	3.41	3.31
L95	3.52	3.42	3.31	3.20	3.10	2.99	2.88	2.77	2.67	2.56	2.45
200	4.58	4.47	4.37	4.26	4.15	4.05	3.94	3.83	3.73	3.62	3.51
L95	3.72	3.62	3.51	3.40	3.30	3.19	3.08	2.97	2.87	2.76	2.65

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 11

FVC in litres for Aboriginal male smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
160	3.21	3.12	3.02	2.92	2.82	2.72	2.62	2.53	2.43	2.33	2.23
L95	2.20	2.10	2.00	1.90	1.81	1.71	1.61	1.51	1.41	1.31	1.20
165	3.48	3.38	3.28	3.18	3.09	2.99	2.89	2.79	2.69	2.60	2.50
L95	2.47	2.37	2.27	2.17	2.08	1.98	1.88	1.78	1.68	1.58	1.48
170	3.74	3.65	3.55	3.45	3.35	3.25	3.16	3.06	2.96	2.86	2.76
L95	2.74	2.64	2.54	2.44	2.34	2.25	2.15	2.05	1.95	1.85	1.74
175	4.01	3.91	3.81	3.72	3.62	3.52	3.42	3.32	3.23	3.13	3.03
L95	3.00	2.90	2.81	2.71	2.61	2.51	2.41	2.31	2.21	2.11	2.01
180	4.28	4.18	4.08	3.98	3.88	3.79	3.69	3.59	3.49	3.39	3.30
L95	3.27	3.17	3.07	2.97	2.88	2.78	2.68	2.58	2.48	2.38	2.27
185	4.54	4.44	4.35	4.25	4.15	4.05	3.95	3.86	3.76	3.66	3.56
L95	3.53	3.43	3.33	3.24	3.14	3.04	2.94	2.84	2.74	2.64	2.54
190	4.81	4.71	4.61	4.51	4.42	4.32	4.22	4.12	4.02	3.93	3.83
L95	3.79	3.69	3.60	3.50	3.40	3.30	3.20	3.10	3.00	2.90	2.80
195	5.07	4.98	4.88	4.78	4.68	4.58	4.49	4.39	4.29	4.19	4.09
L95	4.05	3.95	3.85	3.76	3.66	3.56	3.46	3.36	3.26	3.16	3.06
200	5.34	5.24	5.14	5.05	4.95	4.85	4.75	4.65	4.55	4.46	4.36
L95	4.31	4.21	4.11	4.01	3.91	3.82	3.72	3.62	3.52	3.42	3.31

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 12

FEV_1 in litres for Aboriginal females aged 9-19 years.

	Age										
Ht	9	10	11	12	13	14	15	16	17	18	19
110	0.72	0.74	0.77	0.80	0.83	0.86	0.89	0.92	0.95	0.97	1.00
L95	0.04	0.07	0.09	0.12	0.14	0.16	0.19	0.21	0.23	0.25	0.27
115	0.87	0.90	0.93	0.96	0.99	1.01	1.04	1.07	1.10	1.13	1.16
L95	0.20	0.23	0.25	0.28	0.30	0.33	0.35	0.37	0.39	0.42	0.44
120	1.03	1.05	1.08	1.11	1.14	1.17	1.20	1.23	1.25	1.28	1.31
L95	0.36	0.38	0.41	0.44	0.46	0.49	0.51	0.53	0.56	0.58	0.60
125	1.18	1.21	1.24	1.27	1.29	1.32	1.35	1.38	1.41	1.44	1.47
L95	0.51	0.54	0.57	0.60	0.62	0.65	0.67	0.70	0.72	0.74	0.76
130	1.33	1.36	1.39	1.42	1.45	1.48	1.51	1.54	1.56	1.59	1.62
L95	0.67	0.70	0.73	0.76	0.78	0.81	0.83	0.86	0.88	0.90	0.93
135	1.49	1.52	1.55	1.58	1.60	1.63	1.66	1.69	1.72	1.75	1.78
L95	0.83	0.86	0.88	0.91	0.94	0.97	0.99	1.02	1.04	1.07	1.09
140	1.64	1.67	1.70	1.73	1.76	1.79	1.82	1.84	1.87	1.90	1.93
L95	0.98	1.01	1.04	1.07	1.10	1.12	1.15	1.18	1.20	1.23	1.25
145	1.80	1.83	1.86	1.89	1.91	1.94	1.97	2.00	2.03	2.06	2.09
L95	1.13	1.17	1.20	1.22	1.25	1.28	1.31	1.33	1.36	1.38	1.41
150	1.95	1.98	2.01	2.04	2.07	2.10	2.13	2.15	2.18	2.21	2.24
L95	1.29	1.32	1.35	1.38	1.41	1.44	1.46	1.49	1.52	1.54	1.57
155	2.11	2.14	2.17	2.19	2.22	2.25	2.28	2.31	2.34	2.37	2.40
L95	1.44	1.47	1.50	1.53	1.56	1.59	1.62	1.65	1.67	1.70	1.72
160	2.26	2.29	2.32	2.35	2.38	2.41	2.44	2.46	2.49	2.52	2.55
L95	1.59	1.62	1.65	1.68	1.72	1.74	1.77	1.80	1.83	1.86	1.88
165	2.42	2.45	2.48	2.50	2.53	2.56	2.59	2.62	2.65	2.68	2.70
L95	1.74	1.77	1.80	1.84	1.87	1.90	1.93	1.96	1.98	2.01	2.04
170	2.57	2.60	2.63	2.66	2.69	2.72	2.75	2.77	2.80	2.83	2.86
L95	1.89	1.92	1.95	1.99	2.02	2.05	2.08	2.11	2.14	2.16	2.19
175	2.73	2.76	2.79	2.81	2.84	2.87	2.90	2.93	2.96	2.99	3.01
L95	2.04	2.07	2.10	2.14	2.17	2.20	2.23	2.26	2.29	2.32	2.35
180	2.88	2.91	2.94	2.97	3.00	3.03	3.05	3.08	3.11	3.14	3.17
L95	2.18	2.22	2.25	2.29	2.32	2.35	2.38	2.41	2.44	2.47	2.50
185	3.04	3.07	3.09	3.12	3.15	3.18	3.21	3.24	3.27	3.30	3.32
L95	2.33	2.36	2.40	2.43	2.47	2.50	2.53	2.56	2.59	2.62	2.65

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 13

FVC in litres for Aboriginal females aged 9-19 years.

	Age										
Ht	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**
110	0.78	0.81	0.85	0.88	0.91	0.95	0.98	1.02	1.05	1.09	1.12
L95	0.02	0.05	0.08	0.11	0.14	0.17	0.20	0.23	0.25	0.28	0.30
115	0.94	0.97	1.01	1.04	1.08	1.11	1.14	1.18	1.21	1.25	1.28
L95	0.19	0.22	0.25	0.28	0.31	0.34	0.37	0.40	0.42	0.45	0.48
120	1.10	1.13	1.17	1.20	1.24	1.27	1.30	1.34	1.37	1.41	1.44
L95	0.35	0.39	0.42	0.45	0.48	0.51	0.54	0.57	0.59	0.62	0.65
125	1.26	1.29	1.33	1.36	1.40	1.43	1.47	1.50	1.53	1.57	1.60
L95	0.52	0.55	0.58	0.61	0.65	0.68	0.71	0.73	0.76	0.79	0.82
130	1.42	1.46	1.49	1.52	1.56	1.59	1.63	1.66	1.69	1.73	1.76
L95	0.68	0.71	0.75	0.78	0.81	0.84	0.87	0.90	0.93	0.96	0.99
135	1.58	1.62	1.65	1.68	1.72	1.75	1.79	1.82	1.86	1.89	1.92
L95	0.84	0.88	0.91	0.94	0.98	1.01	1.04	1.07	1.10	1.13	1.15
140	1.74	1.78	1.81	1.85	1.88	1.91	1.95	1.98	2.02	2.05	2.08
L95	1.00	1.04	1.07	1.11	1.14	1.17	1.20	1.23	1.26	1.29	1.32
145	1.90	1.94	1.97	2.01	2.04	2.07	2.11	2.14	2.18	2.21	2.25
L95	1.16	1.20	1.23	1.27	1.30	1.33	1.37	1.40	1.43	1.46	1.49
150	2.06	2.10	2.13	2.17	2.20	2.24	2.27	2.30	2.34	2.37	2.41
L95	1.32	1.36	1.39	1.43	1.46	1.50	1.53	1.56	1.59	1.62	1.65
155	2.22	2.26	2.29	2.33	2.36	2.40	2.43	2.46	2.50	2.53	2.57
L95	1.48	1.51	1.55	1.59	1.62	1.66	1.69	1.72	1.76	1.79	1.82
160	2.39	2.42	2.45	2.49	2.52	2.56	2.59	2.63	2.66	2.69	2.73
L95	1.63	1.67	1.71	1.74	1.78	1.82	1.85	1.88	1.92	1.95	1.98
165	2.55	2.58	2.61	2.65	2.68	2.72	2.75	2.79	2.82	2.85	2.89
L95	1.79	1.83	1.86	1.90	1.94	1.97	2.01	2.04	2.08	2.11	2.14
170	2.71	2.74	2.78	2.81	2.84	2.88	2.91	2.95	2.98	3.02	3.05
L95	1.94	1.98	2.02	2.06	2.10	2.13	2.17	2.20	2.24	2.27	2.30
175	2.87	2.90	2.94	2.97	3.00	3.04	3.07	3.11	3.14	3.18	3.21
L95	2.09	2.13	2.17	2.21	2.25	2.29	2.33	2.36	2.40	2.43	2.46
180	3.03	3.06	3.10	3.13	3.17	3.20	3.23	3.27	3.30	3.34	3.37
L95	2.25	2.29	2.33	2.37	2.41	2.44	2.48	2.52	2.55	2.59	2.62
185	3.19	3.22	3.26	3.29	3.33	3.36	3.39	3.43	3.46	3.50	3.53
L95	2.40	2.44	2.48	2.52	2.56	2.60	2.64	2.67	2.71	2.74	2.78

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 14

FEV_1 in litres for Aboriginal female non-smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
140	1.71	1.60	1.50	1.39	1.28	1.18	1.07	0.96	0.86	0.75	0.64
L95	0.86	0.76	0.65	0.54	0.44	0.33	0.22	0.11	0.01	0.00	0.00
145	1.92	1.81	1.71	1.60	1.49	1.39	1.28	1.17	1.07	0.96	0.85
L95	1.08	0.97	0.86	0.76	0.65	0.54	0.44	0.33	0.22	0.11	0.00
150	2.13	2.02	1.91	1.81	1.70	1.59	1.49	1.38	1.27	1.17	1.06
L95	1.29	1.84	1.08	0.97	0.86	0.76	0.65	0.54	0.43	0.32	0.22
155	2.34	2.23	2.12	2.02	1.91	1.80	1.70	1.59	1.48	1.38	1.27
L95	1.50	1.39	1.29	1.18	1.08	0.97	0.86	0.75	0.64	0.54	0.43
160	2.54	2.44	2.33	2.22	2.12	2.01	1.90	1.80	1.69	1.58	1.48
L95	1.71	1.60	1.50	1.39	1.29	1.18	1.07	0.96	0.85	0.75	0.64
165	2.75	2.65	2.54	2.43	2.33	2.22	2.11	2.01	1.90	1.79	1.69
L95	1.92	1.81	1.71	1.60	1.49	1.39	1.28	1.17	1.06	0.95	0.84
170	2.96	2.85	2.75	2.64	2.53	2.43	2.32	2.21	2.11	2.00	1.89
L95	2.12	2.02	1.91	1.81	1.70	1.59	1.48	1.38	1.27	1.16	1.05
175	3.17	3.06	2.96	2.85	2.74	2.64	2.53	2.42	2.32	2.21	2.10
L95	2.33	2.22	2.12	2.01	1.90	1.80	1.69	1.58	1.47	1.36	1.25
180	3.38	3.27	3.16	3.06	2.95	2.84	2.74	2.63	2.52	2.42	2.31
L95	2.53	2.43	2.32	2.21	2.11	2.00	1.89	1.78	1.68	1.57	1.46

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 15

FVC in litres for Aboriginal female non-smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
140	1.65	1.55	1.45	1.36	1.26	1.16	1.06	0.96	0.87	0.77	0.67
L95	0.63	0.53	0.43	0.34	0.24	0.14	0.04	0.00	0.00	0.00	0.00
145	1.92	1.82	1.72	1.62	1.52	1.43	1.33	1.23	1.13	1.03	0.94
L95	0.90	0.80	0.71	0.61	0.51	0.41	0.31	0.21	0.11	0.01	0.00
150	2.18	2.08	1.99	1.89	1.79	1.69	1.59	1.50	1.40	1.30	1.20
L95	1.17	1.07	0.98	0.88	0.78	0.68	0.58	0.48	0.38	0.28	0.18
155	2.45	2.35	2.25	2.15	2.06	1.96	1.86	1.76	1.66	1.57	1.47
L95	1.44	1.34	1.25	1.15	1.05	0.95	0.85	0.75	0.65	0.55	0.45
160	2.71	2.62	2.52	2.42	2.32	2.22	2.13	2.03	1.93	1.83	1.73
L95	1.71	1.61	1.51	1.42	1.32	1.22	1.12	1.02	0.92	0.82	0.72
165	2.98	2.88	2.78	2.69	2.59	2.49	2.39	2.29	2.20	2.10	2.00
L95	1.97	1.87	1.78	1.68	1.58	1.48	1.38	1.28	1.18	1.08	0.98
170	3.25	3.15	3.05	2.95	2.85	2.76	2.66	2.56	2.46	2.36	2.26
L95	2.23	2.14	2.04	1.94	1.85	1.75	1.65	1.55	1.45	1.35	1.25
175	3.51	3.41	3.32	3.22	3.12	3.02	2.92	2.82	2.73	2.63	2.53
L95	2.50	2.40	2.30	2.20	2.11	2.01	1.91	1.81	1.71	1.61	1.51
180	3.78	3.68	3.58	3.48	3.38	3.29	3.19	3.09	2.99	2.89	2.80
L95	2.75	2.66	2.56	2.46	2.37	2.27	2.17	2.07	1.97	1.87	1.77

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 16

FEV_1 in litres for Aboriginal female smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
140	1.85	1.74	1.63	1.53	1.42	1.31	1.21	1.10	0.99	0.89	0.78
L95	1.00	0.89	0.78	0.68	0.57	0.46	0.35	0.25	0.14	0.03	0.00
145	2.06	1.95	1.84	1.74	1.63	1.52	1.42	1.31	1.20	1.10	0.99
L95	1.21	1.11	1.00	0.89	0.78	0.68	0.57	0.46	0.35	0.24	0.13
150	2.27	2.16	2.05	1.95	1.84	1.73	1.63	1.52	1.41	1.30	1.20
L95	1.43	1.32	1.21	1.11	1.00	0.89	0.78	0.67	0.56	0.45	0.34
155	2.47	2.37	2.26	2.15	2.05	1.94	1.83	1.73	1.62	1.51	1.41
L95	1.64	1.53	1.42	1.32	1.21	1.10	0.99	0.88	0.78	0.67	0.56
160	2.68	2.58	2.47	2.36	2.26	2.15	2.04	1.94	1.83	1.72	1.62
L95	1.85	1.74	1.63	1.53	1.42	1.31	1.20	1.09	0.99	0.88	0.77
165	2.89	2.78	2.68	2.57	2.46	2.36	2.25	2.14	2.04	1.93	1.82
L95	2.05	1.95	1.84	1.74	1.63	1.52	1.41	1.30	1.19	1.08	0.97
170	3.10	2.99	2.89	2.78	2.67	2.57	2.46	2.35	2.25	2.14	2.03
L95	2.26	2.16	2.05	1.94	1.83	1.73	1.62	1.51	1.40	1.29	1.18
175	3.31	3.20	3.09	2.99	2.88	2.77	2.67	2.56	2.45	2.35	2.24
L95	2.47	2.36	2.25	2.15	2.04	1.93	1.82	1.71	1.61	1.50	1.39
180	3.52	3.41	3.30	3.20	3.09	2.98	2.88	2.77	2.66	2.56	2.45
L95	2.67	2.56	2.46	2.35	2.24	2.13	2.03	1.92	1.81	1.70	1.59

L95= lower limit of the 95% confidence interval around the estimate.

Appendix 17

FVC in litres for Aboriginal female smokers aged 20-70 years.

	Age										
Ht	20	25	30	35	40	45	50	55	60	65	70
140	1.91	1.81	1.72	1.62	1.52	1.42	1.32	1.22	1.13	1.03	0.93
L95	0.88	0.79	0.69	0.59	0.49	0.39	0.29	0.19	0.09	0.00	0.00
145	2.18	2.08	1.98	1.88	1.78	1.69	1.59	1.49	1.39	1.29	1.20
L95	1.16	1.06	0.96	0.86	0.76	0.67	0.57	0.46	0.36	0.26	0.16
150	2.44	2.34	2.25	2.15	2.05	1.95	1.85	1.76	1.66	1.56	1.46
L95	1.43	1.33	1.23	1.14	1.04	0.94	0.84	0.74	0.64	0.53	0.43
155	2.71	2.61	2.51	2.41	2.32	2.22	2.12	2.02	1.92	1.83	1.73
L95	1.70	1.60	1.50	1.40	1.31	1.21	1.11	1.01	0.90	0.80	0.70
160	2.97	2.88	2.78	2.68	2.58	2.48	2.39	2.29	2.19	2.09	1.99
L95	1.97	1.87	1.77	1.67	1.57	1.47	1.37	1.27	1.17	1.07	0.97
165	3.24	3.14	3.04	2.95	2.85	2.75	2.65	2.55	2.46	2.36	2.26
L95	2.23	2.13	2.04	1.94	1.84	1.74	1.64	1.54	1.44	1.34	1.23
170	3.51	3.41	3.31	3.21	3.11	3.02	2.92	2.82	2.72	2.62	2.53
L95	2.50	2.40	2.30	2.20	2.10	2.00	1.90	1.80	1.70	1.60	1.50
175	3.77	3.67	3.58	3.48	3.38	3.28	3.18	3.09	2.99	2.89	2.79
L95	2.76	2.66	2.56	2.46	2.36	2.26	2.16	2.06	1.96	1.86	1.76
180	4.04	3.94	3.84	3.74	3.65	3.55	3.45	3.35	3.25	3.15	3.06
L95	3.02	2.92	2.82	2.72	2.62	2.52	2.42	2.32	2.22	2.12	2.02

L95= lower limit of the 95% confidence interval around the estimate.